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H. Preston Hoskins, Secretary-Editor, 1230 W. Washington Blvd., Chicago, Ill.

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HATS OFF TO ATLANTA

Depression or no depression, the veterinarians of the southern states determined to make the 1932 convention of the A. V. M. A. something to be remembered. They came through with flying colors. Atlanta will remain in the minds of the 600 who were present as a synonym of southern hospitality. Although the attendance was below the average of recent years, the meeting was outstanding in a number of respects. The literary program exceeded those of all previous meetings in the number of papers presented. The clinic was exceptionally well arranged and provided a wealth of material. The banquet held in the open air, on the veranda of the Atlanta Biltmore, was unique as far as the setting was concerned, and at the same time highly enjoyable by reason of the brief, sparkling speeches, the fine musical entertainment and the pleasant weather conditions. There was enough time left, after the banquet, for dancing and bridge.

Monday, the day before the opening session, saw several hundred registered. Meetings of the Executive Board and various committees of the A. V. M. A., as well as the meeting of the Southern States Veterinary Medical Association, attracted the heavy advance guard. The opening session Tuesday morning saw the Georgian Ballroom at the Atlanta Biltmore comfortably filled with an audience that was just about evenly divided be-

tween the sexes. One of the high spots of the meeting was the unusually large proportion of the fair sex in attendance. Unofficial figures give the number of ladies as about 200. President Dykstra's address, published in this issue of the JOURNAL, was received with eager attention, and later referred to a special committee for study. This committee will report to the Executive Board at the earliest possible date.

The business sessions went off with clock-like precision. President Dykstra called each session to order at the appointed time. Every report was ready when called for, and most of them were brief and snappy. As a result the business sessions did not drag. Each afternoon an adjournment was possible early, leaving an hour or more for "visiting" before dinner. The sectional meetings were well attended and the papers and discussions took all the time that was available. Several of the papers were read by title, due to the inability of the authors to be present. All of these papers, of course, will be published in the JOURNAL. The Section on Military Medicine functioned for the first time and drew a fair-sized attendance of veterinarians interested in military matters.

The election of officers brought out no contest. Dr. N. F. Williams, Texas State Veterinarian, was the unanimous choice for the office of president. His election creates a vacancy on the Executive Board for District 8, which will be filled temporarily by Dr. J. C. Flynn, of Kansas City, Mo. An election will be held in District 8, for the purpose of selecting a Board member to serve for the balance of Dr. Williams' unexpired term (until 1936).

The five vice-presidents were elected unanimously as follows:

First Vice-President—Colonel Robert J. Foster, V. C., U. S. Army, Fort Bliss, Texas.

Second Vice-President—Dr. George C. Faville, State Division of Animal Industry, Richmond, Va.

Third Vice-President—Dr. W. A. Axby, Practitioner, Harrison, O.

Fourth Vice-President—Dr. J. A. Campbell, Practitioner, Toronto, Ontario, Canada.

Fifth Vice-President—Dr. George Alexander Dick, University of Pennsylvania, Philadelphia, Pa.

Dr. M. Jacob, of Knoxville, Tenn., will continue as custodian of our funds for another year (his sixteenth consecutive term as A. V. M. A. treasurer). Dr. Robert S. MacKellar, well-known practitioner of New York City, was reëlected member-at-large

of the Executive Board for another five-year term, and that body promptly selected Dr. MacKellar for the office of Chairman for another year. Dr. A. E. Cameron succeeded Dr. George Hilton as member of the Executive Board for District 1, at the close of the meeting, and Dr. H. W. Jakeman, of Boston, Mass., succeeded Dr. D. H. Udall, in District 9.

The selection of a meeting place for 1933 was referred to the Executive Board, after invitations had been received from Chicago, Toronto and Mexico. The Board recommended Chicago and this action subseqeuntly was approved by the Association, which body also approved a recommendation of the Board that the 1934 meeting be held in New York City, rather than Philadelphia, as previously planned. Certain matters in connection with the International Veterinary Congress suggested the advisability of the change.

For the first time in several years, an honorary member was added to the roll. This honor was conferred upon Dr. C. N. McBryde, of the U. S. Bureau of Animal Industry, in recognition of his contributions to veterinary medicine. Dr. McBryde read two very interesting papers at the meeting and was able to receive official notification of his election in person, right at the meeting—a rare occasion.

The Committee on Local Arrangements received the well-earned thanks of every one of the 600 in attendance. Never has a local committee labored under greater handicaps in preparing for an A. V. M. A. convention. The veterinary population of Atlanta is smaller than that of most cities in which we have met recently and none of the local men had had any previous experience in planning for an A. V. M. A. meeting. The territory tributary to Atlanta is not heavily populated with veterinarians and the natural result was that a comparatively small group had to carry the load. The economic situation made it hard to secure funds for entertainment, which added to the difficulties. However, the committee came through in fine style and richly deserved every bit of the praise which the visitors so willingly gave them. No one left Atlanta without an opportunity to know just what is meant by southern hospitality, and every one will look forward to a return visit at some time in the future.

The full proceedings of the sixty-ninth annual convention will be published in the October issue of the JOURNAL. In this brief preliminary report, there has been no opportunity to comment on the real business of the convention. There was a lot of it.

Read it carefully next month. Only in this way can you keep tab on what the A. V. M. A. is doing.

Remember, Chicago in 1933.

Convention Notes

Georgia led the states in attendance, with 133 registrations.

Dr. B. T. Simms, of Corvallis, Ore., kept up his unbroken record of attendance.

Dr. C. C. Palmer, of Newark, Del., was the only member present from the Blue Hen State.

Dr. H. W. Jakeman, of Boston, and Dr. W. H. Dodge, of Leominster, represented the Old Bay State.

All states east of the Mississippi, except Vermont, Rhode Island, Connecticut and New Jersey, were represented.

Ohio contributed almost ten per cent of the attendance, with 59 Buckeyes present.

Dr. J. H. Bux, of Little Rock, Ark., was the only member from the Bear State at the meeting.

Dr. J. C. McGrath, of Phoenix, Ariz., was the sole representative of the profession from Arizona.

Dr. A. L. Edmunds, of Franklin, N. H., was the only member present from the Granite State.

Dr. R. W. Hixson, of Falls City, Neb., was the only Nebraskan at the meeting.

Drs. S. E. Hershey and H. M. Newton, of Charleston, upheld the honor of West Virginia.

Dr. Francisco Moguel M., attaché of the Mexican Embassy, enjoyed his first A. V. M. A. meeting.

Dr. C. W. Crowley, of Saint Louis, Mo., who joined the A. V. M. A. in 1876, was the oldest member in attendance.

Dr. J. Franklin Witter, of Orono, was Maine's only veterinarian at the convention.

Dr. Aubrey M. Lee, of Laramie, Wyo., was one of the "long-distance" members at the convention.

Dr. W. F. Crewe, of Bismarck, N. Dak., represented his district at the Executive Board meetings, in the absence of Dr. C. H. Hays.

A trio of veterinarians were registered from the Lone Star Fitch, Saint Paul; C. F. Schlotthauer, Rochester, and R. L. West, Waseca.

Michigan had five members on the registration list: Drs. E. T.

Hallman and J. W. Patton, East Lansing; A. S. Schlingman, Detroit; J. W. Schneider, Riga, and F. E. Stiles, Battle Creek.

A quintet of Maryland veterinarians registered: Drs. W. R. Crawford, Westminster; Chas. R. Davis and E. M. Pickens, College Park; E. B. Dibbell, Baltimore; G. A. Edmiston, Easton.

Dr. Tait Butler, of Memphis, Tenn., with 45 years of continuous membership to his credit, was among the "old-timers" who were at the meeting.

Six Kansas members represented the Sunflower State: Drs. C. W. Bower, Topeka; R. R. Dykstra and E. J. Frick, Manhattan; T. J. Leasure, Lawrence; M. P. Schlaegel, Burr Oak; S. L. Stewart, Olathe.

Nine ex-presidents of the A. V. M. A. were among those present: Drs. Tait Butler, J. R. Mohler, C. J. Marshall, C. A. Cary, A. T. Kinsley, W. H. Welch, C. H. Stange, T. A. Sigler and T. E. Munce.

Seven veterinary colleges were represented by their deans: Brumley, of Ohio; Cary, of Alabama; Dick, of Pennsylvania; Dykstra, of Kansas; Hagan, of Cornell; Richardson, of Georgia; Stange, of Iowa.

Official Washington was represented by Drs. J. R. Mohler, W. M. Mohler, A. E. Wight and W. H. Wright, of the Bureau of Animal Industry; Major R. A. Kelser, of the U. S. Army, and J. E. Shillinger, of the Bureau of Biological Survey.

Iowa sent nine veterinarians to the meeting: Drs. H. D. Bergman, H. H. Dukes, T. S. Leith, F. D. Patterson and C. H. Stange, Ames; A. H. Quin, Jr., Des Moines; H. J. Shore, Fort Dodge; W. C. Vollstedt, Dixon; C. W. Wiley, Farson.

The Keystone State had eleven members in attendance: Drs. E. P. Althouse, Sunbury; G. A. Dick, Henry E. Hess, Wm. H. Ivens, Wm. J. Lee, C. J. Marshall, F. J. Olbrich and E. L. Stubbs, Philadelphia; G. M. Leighow, Danville; T. E. Munce, Harrisburg; John Reichel, Glenolden.

Mississippi had nine veterinarians on the registry: Drs. T. W. Boman, E. H. Durr, Hartwell Robbins, C. E. O'Neal and R. H. Stewart, Jackson; L. L. Denson, Jr., Kosciusko; J. W. Duckworth, Picayune; W. L. Gates, Clarksdale; J. S. Kamper, McComb.

The convention photograph proved to be a flop. After working for almost half an hour to get the crowd in position and everybody looking pleasant, two exposures were made—both on the same plate!

Missouri members to the number of eight helped the attendance figure: Drs. C. W. Crowley and S. W. Haigler, Saint Louis; Hugh E. Curry, G. G. Graham, A. T. Kinsley and Ashe Lockhart, Kansas City; W. C. Dillard, Farmington; J. B. Latshaw, Caruthersville.

Hoosiers to the number of nine made the journey to Atlanta: Drs. R. C. Julien and F. J. Muecke, Indianapolis; W. C. Kortenber, New Haven; H. J. Magrane, Mishawaka; G. W. Musselman, Denver; E. R. Page, Corydon; W. L. Platter and R. E. Wood, Rockville; T. A. Sigler, Greencastle.

Virginia did well to have fourteen veterinarians in attendance: Drs. R. Marshall Codd, Portsmouth; H. C. Cram, H. T. Farmer, Geo. C. Faville and H. C. Givens, Richmond; W. T. Gilchrist, Norfolk; Page M. Graves, Culpeper; Verne C. Hill, Fort Monroe; R. L. Humphrey, Mountsville; E. P. Johnson, L. E. Starr and I. D. Wilson, Blacksburg; A. W. Miller and E. J. Wills, Harrisonburg.

Illinois was not superstitious with thirteen members registered: Drs. S. D. Buzzard, Stewardson; E. A. Cahill, D. M. Campbell and H. Preston Hoskins, Chicago; D. A. Eastman, Moline; J. V. Lacroix, Evanston; N. S. Mayo, Highland Park; L. N. Morin, McLean; James Smellie, Eureka; E. E. Sweebe, Waukegan; Frank Thorp, Jr., Urbana; C. S. Watt, Collinsville; W. H. Welch, Lexington.

Sixteen Tennessee veterinarians took advantage of the proximity of Atlanta to attend the meeting: Drs. Wm. M. Bell, H. L. Fry, J. M. Jones and O. B. Neely, Nashville; J. W. Perry, Pulaski; Tait Butler, John H. Gillmann and E. B. Mount, Memphis; D. Coughlin, M. Jacob and G. H. Woolfolk, Knoxville; W. H. Emig, G. P. Hatchett and F. W. Morgan, Chattanooga; A. L. Rubin, Fountain City; R. L. Whitaker, Fayetteville.

Officers of Women's Auxiliary

The new officers of the Women's Auxiliary to the A. V. M. A. are as follows: Mrs. T. H. Ferguson, president, Lake Geneva, Wis.; Mrs. R. A. Kelser, 1st vice-president, Washington, D. C.; Mrs. R. P. Marsteller, 2nd vice-president, College Station, Tex.; Mrs. C. W. Bower, 3rd vice-president, Topeka, Kan.; Mrs. Wm. Henry Kelly, 4th vice-president, Albany, N. Y.; Mrs. G. G. Graham, Loan Fund secretary, 812 W. 59th Terrace, Kansas City, Mo.; Mrs. S. E. Hershey, secretary, Box 283, Charleston, W. Va.; Mrs. H. Preston Hoskins, treasurer, 2766 Garrison Ave., Evanston, Ill.

Medals Awarded

The Southern States A. V. M. A. Committee honored the four oldest members of the Association by awarding each one a beautiful medal appropriately inscribed. Only one of the four was present, Dr. C. W. Crowley, of Saint Louis, Mo., who joined in 1876. He was presented with his medal at the banquet, President-elect Williams doing the honors. The other recipients of this rare honor were Dr. J. C. Meyer, of Cincinnati, Ohio, who joined in 1875; Dr. Benjamin McInnes, of Charleston, S. C., who joined one year later, with Dr. Crowley, and Dr. Lester H. Howard, of Brookline, Mass., the oldest living ex-vice-president of the A. V. M. A., who rounded out fifty years of membership this year.

Honorary Degree for Doctor Smith

Dr. Theobald Smith, of the Rockefeller Institute for Medical Research, recently received the honorary degree, doctor of veterinary medicine, from the University of Giessen, Germany. The occasion was the hundredth anniversary of the granting of veterinary degrees at the University.

APPLICATIONS FOR MEMBERSHIP

(See July, 1932, JOURNAL)

FIRST LISTING

ALDRICH, PERCY M. Dixie, Wash.
B. S., D. V. M., Washington State College, 1932
Vouchers: G. W. McNutt and J. R. Fuller.

BENNETT, LESLIE H. Box 688, Monroe, La.
D. V. M., Ohio State University, 1923
Vouchers: H. H. Baur and John H. Gillmann.

BUCKINGHAM, DAVID E. 2115-14th St. N. W., Washington, D. C.
V. M. D., University of Pennsylvania, 1893
Vouchers: H. E. Moskey and L. T. Giltner.

CARLISLE, BILLY E. Camilla, Ga.
D. V. M., Alabama Polytechnic Institute, 1917
Vouchers: E. D. King, Jr. and C. A. Cary.

HOGAN, JAMES H. 517 Miller Ave., South San Francisco, Calif.
M. D. V., McKillip Veterinary College, 1909
Vouchers: S. R. Parker and L. Bilikam.

LOCKE, HARRY A. 1422 Irving St. N. W., Washington, D. C.
D. V. M., George Washington University, 1912
Vouchers: H. E. Moskey and L. T. Giltner.

NATHAN, SIMEON A. 410 Pittsboro St., Chapel Hill, N. C.
D. V. M., Kansas City Veterinary College, 1916
Vouchers: Wm. Moore and L. J. Faulhaber.

RUBIN, ALFRED L. Fountain City, Tenn.
D. V. M., Ohio State University, 1918
Vouchers: D. Coughlin and O. V. Brumley.

SEYMOUR, WALTER E. 2115-14th St. N. W., Washington, D. C.
D. V. M., George Washington University, 1918
Vouchers: H. E. Moskey and L. T. Giltner.

SNELLING, ALBERT M. 198 Hull St., Athens, Ga.
D. V. M., Cornell University, 1931
Vouchers: J. E. Severin and J. L. Hopping.

STEWART, RUSSEL D. Wren, Ohio
D. V. M., Ohio State University, 1917
Vouchers: J. N. Shoemaker and W. F. Guard.

TAYLOR, ROBERT E. Hendersonville, N. C.
D. V. M., Cincinnati Veterinary College, 1916
Vouchers: Harry Gieskemeyer and Wm. Moore.

WEADON, F. MASON, JR. 2115-14th St. N. W., Washington, D. C.
V. M. D., University of Pennsylvania, 1922
Vouchers: H. E. Moskey and L. T. Giltner.

WILLIAMSON, ARTHUR H. c/o Health Dept., Charlotte, N. C.
B. S., D. V. M., Alabama Polytechnic Institute, 1924
Vouchers: H. Calvin Rea and S. W. Haigler.

WITTER, J. FRANKLIN
Dept. of Animal Industry, University of Maine, Orono, Me.
B. S., D. V. M., Michigan State College, 1932
Vouchers: E. T. Hallman and E. P. Johnson.

Applications Pending

SECOND LISTING

(See August, 1932, JOURNAL)

Almquist, Elvin W., Box 212, R. 1, Beaverton, Ore.
Bendix, Wilmer L., Box 100, Dumbarton, Va.
Campbell, Daniel L., 820 N. E. Third Ave., Fort Lauderdale, Fla.
Craige, A. Henry, 1317 S. 57th St., Philadelphia, Pa.
DeCamp, Clayton E., c/o Rare Chemicals, Inc., Gray Oaks Ave., Nepera Park, N. Y.
Huff, Joseph Neal, 4950 York St., Denver, Colo.
King, Joseph J., 1100 N. St., Sacramento, Calif.
Mills, Henry Lee, 339 Colchester Ave., Burlington, Vt.
O'Connell, John A., Room 1205, 11 Beacon St., Boston, Mass.
Perry, Lucius D., 20 Ferris St., Saint Albans, Vt.
Rumney, Wilfred J., 612 King St. W., Hamilton, Ont., Can.

The amount which should accompany an application filed this month is $6.67, which covers membership fee and dues to January 1, 1933, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

Eastern Iowa Veterinary Association. Clinic. Mechanicsville, Iowa. September 6, 1932. Dr. Iva Dunn, Secretary, Atkins, Iowa.

New York City, Veterinary Medical Association of. Academy of Medicine, 5th Ave. & 103rd St., New York, N. Y. September 7, 1932. Dr. John E. Crawford, Secretary, 708 Beach 19th St., Far Rockaway, Long Island, N. Y.

San Diego-Imperial Veterinary Medical Association. San Diego, Calif. September 7, 1932. Dr. A. P. Immenschuh, Secretary, Santee, Calif.

South Eastern Wisconsin Veterinary Association. Kewaskum, Wis. September 8, 1932. Dr. J. O. McCoy, Secretary, Reeseville, Wis.

Tulsa County Veterinary Association. Tulsa, Okla. September 8, 1932. Dr. J. M. Higgins, Secretary, 3305 E. 11th St., Tulsa, Okla.

Interstate Veterinary Medical Association. Elks Building, Omaha, Neb. September 12, 1932. Dr. G. L. Taylor, Secretary, Plattsmouth, Neb.

Chicago Veterinary Medical Association. Atlantic Hotel, Chicago, Ill. September 13, 1932. Dr. E. E. Sweebe, Secretary, 14th St. & Sheridan Road, North Chicago, Ill.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. September 14, 1932. Dr. A. S. Schlingman, Secretary, Parke, Davis & Co., Detroit, Mich.

Kansas City Veterinary Association. Baltimore Hotel, Kansas City, Mo. September 20, 1932. Dr. J. D. Ray, Secretary, 1103 E. 47th St., Kansas City, Mo.

Southern California Veterinary Medical Association. Chamber of Commerce Bldg., Los Angeles, Calif. September 21, 1932. Dr. E. E. Jones, Secretary, 1451 Mirasol St., Los Angeles, Calif.

Utah Veterinary Medical Association. State Capitol Building, Salt Lake City, Utah. October 6, 1932. Dr. E. A. Bundy, Secretary, 1656 25th St., Ogden, Utah.

Eastern Iowa Veterinary Association. Hotel Montrose, Cedar Rapids, Iowa. October 11-12, 1932. Dr. Iva Dunn, Secretary, Atkins, Iowa.

Maine Veterinary Medical Association. State House, Augusta, Me. October 12, 1932. Dr. L. E. Maddocks, Secretary, R. F. D. 2, Augusta, Me.

The American Humane Association. San Francisco, Calif. October 18-20, 1932. J. N. Walker, General Manager, 80 Howard, New York, N. Y.

Pennsylvania State Veterinary Medical Association. Pennsylvania State College, State College, Pa. October 20-21, 1932. Dr. Thos. D. James, Secretary, 816 N. Main St., Scranton, Pa.

American Public Health Association, The. Willard Hotel, Washington, D. C. October 24-27, 1932. Willimina Rayne Walsh, Secretary, 450 Seventh Ave., New York, N. Y.

ADDRESS OF THE PRESIDENT*

By R. R. DYKSTRA, *President,*
American Veterinary Medical Association,
Manhattan, Kan.

I. INTRODUCTORY AND ACKNOWLEDGMENTS

At each annual meeting of the American Veterinary Medical Association the President, who presumably has had a year's time to study the various problems concerning the profession, is called upon to place his deductions and recommendations before the membership of the Association. In the past, the Association has had delivered to it many notable presidential addresses, though none excels the one delivered a year ago in Kansas City by the immediate past president. It contains so many pertinent, carefully weighed suggestions and recommendations that the presidential address this year would virtually be a complete one, were no more said than that a reaffirmation of the principles laid down at that time is our contribution this year. So commendable was the presidential address of a year ago that a special committee was appointed to study it thoroughly, and after having digested it, to make recommendations to the Executive Board and the membership. The personnel of the committee, I am certain, has devoted much time to the duty assigned it and the Association will profit accordingly.

1. A president elected for a period of one year, and without specific knowledge as to the workings of the organization and its numerous ramifications, would be hopelessly lost did he not have as a constant source of information, advice and encouragement, the Secretary, to fall back upon. In many of the difficulties encountered by us we have found his judgment good, his knowledge of veterinary affairs intimate, and his actions at all times courteous and helpful.

2. In the American Veterinary Medical Association, as in all other organizations having a large membership, the real work is done in the various committee rooms. Your President feels that he is under the greatest obligations to the large number of veterinarians in all parts of the United States who have come enthusiastically to his assistance when they were requested to assume some professional obligation. Only those who have

*Presented at the sixty-ninth annual meeting of the American Veterinary Medical Association, Atlanta, Ga., August 23-26, 1932.

served in this capacity, so that they may speak from personal experience, and those who are in executive positions, so that they may have an opportunity to observe the results obtained, can realize to the fullest extent the self-sacrificing spirit and the love of the profession that has actuated these men to give freely of their time, energies and money for the advancement of the organization and the profession.

3. Organizations as a whole are judged very largely by the professional attitude of those members, either as individuals or in groups, who are more or less in the public eye. The veterinary profession is particularly fortunate in having several groups of this character, and I cannot forego the opportunity to say a brief word of commendation in regard to each of them.

(a) Possibly the group of veterinarians having the most frequent and direct contact with the public is that group in the United States Bureau of Animal Industry, in the Dominion Health of Animals Branch, and the state veterinarians and their staffs. Careful readers of the daily press and of the various trade journals, which frequently contain expressions from live stock owners, cannot fail to be impressed by the almost universally favorable opinion that the work of this group has engendered. The veterinary profession has been placed on a higher professional plane by the general public, because of the thoroughly professional manner in which they handle their numerous and arduous duties.

(b) In a like manner, officers of the Veterinary Corps in the United States Army, though their contacts are probably not so numerous, have by their very high attainments, and by their gentlemanly and professional deportment, created for the veterinary profession an atmosphere of learning and gentility that is causing, in circles where it will do the most good, a favorable reaction for the profession that could be obtained in no other manner.

(c) In its professional commercial houses the veterinary profession also has been unusually fortunate. Your President can speak from personal knowledge in regard to this group. They are genuinely interested in advancing not only the professional, but the material interests of the veterinarian, and they are doing it in a manner that reflects distinct credit upon the profession as a whole. It is not difficult to select these highly ethical firms and those in a position to do so should encourage them in every manner possible, and at the same time discourage those that are showing a total lack of interest

in the veterinary profession, and incidentally in those served by the veterinarian. A commercial supply house so short-sighted that it will cater to the professionally untrained animal-owner, and disregard those trained to give a high measure of service, should not have the financial or scientific encouragement of the members of this group. It is axiomatic that only those best trained in animal disease problems are qualified to give the best service, and to disregard these can only lead to dire results for the live stock owners. This organization, therefore, is on firm and proven ground when it encourages those in harmony with this axiom and discourages those that are not following its principles.

(d) I have intentionally left for the last the private practitioner. During the year now almost closed, it has been the privilege of your President to have a very large number of contacts with practicing veterinarians in all parts of the United States. We have found them uniformly courteous, gentlemanly, and professionally inquisitive to a degree seldom found in those who have been away from the academic halls of learning for a greater or less period of time. When members of a profession are so insistent upon maintaining their professional knowledge upon as high a level as veterinarians are, it speaks well for the profession. The veterinary profession is judged very largely by its representative in a local community. When this is multiplied by the large number of units in United States and Canada served by men of this type, we then have good reasons for the high plane upon which the profession now rests.

4. No presidential address can be complete were there not also a word of admonishment regarding some weaknesses. We have at times been grieved to note that there did not exist that close social, business and professional coöperation that should be present between the various parts of our organization. It is professionally malicious and unsound for those in the same field of human endeavor to engage in bickerings, accusations and counteraccusations, or in any way, either by words or actions, to reflect upon the integrity of another in the same line of endeavor. Professions closely related to veterinary medicine long ago learned that they must present a solid front if they wish to influence public opinion favorably. The result in these organizations has been that, as far as the general public at least is concerned, they are "air-tight," and it is virtually impossible to find any single member who will make derogatory remarks in the

presence of a layman about a professional brother. In concluding this paragraph we want again to stress that, in lay affairs, an attitude of petty maliciousness is to be condemned, and doubly so in professional life.

5. Possibly it is not amiss also to mention here one of the tribulations of the President of the American Veterinary Medical Association. I am referring to the fact that members of this organization, in their enthusiasm for definite objectives, make certain recommendations on the floor during the general sessions, though these same members seem to lack a continuing enthusiasm, because when at a later period they are asked to give the Association the benefit of their wisdom and experience, they are not willing to assume a responsibility as a member of the very committee that is being formed to take charge of the activity originally sponsored on the floor. Your President feels that if any member of this organization makes a "case" before the membership, he should be willing to guide the future destinies of his child.

II. Recommendations

Possibly it is the financially strenuous period through which we are passing that is flavoring the address of your President this year.

During the past year, most veterinarians have been sorely tried, as have others, by conditions entirely beyond their control. For veterinarians the situation was accentuated, because during the decade at least there was injected into their financial picture a most determined and terrific effort to mechanize American farms, "to displace an agricultural product made on the farm, and raised and maintained on other farm products." Their behavior and service during this trial justifies our pride in being a part of this profession. If there ever has been a doubt in the minds of any concerning the future of the veterinary profession, its accomplishments in the face of adversity should dispel this forever.

Having in mind, however, the conditions through which the profession has just passed, your President feels that if he can make some suggestions that in the future may assist in warding off, to some extent, at least, the influences of an unfavorable financial situation, he will have contributed his small part.

The purposes of the American Veterinary Medical Association, as stated in Article II of the Constitution, are as follows:

a. To protect and promote the professional interests of the veterinarian.

b. To elevate the standard of veterinary education.
c. To procure uniform laws and regulations governing veterinary practice, and the control of diseases of animals, including poultry.
d. To direct public opinion regarding problems of animals, including poultry hygiene.
e. To promote good fellowship in the profession.

No better maxims for the guidance of an association can be laid down anywhere, but we should maintain a proper balance between them, and at times a greater stress should be placed upon certain phases of it. As an example of this, one of our state associations a few years ago stated in its objects the following:

"To protect the material interests of the veterinary profession * * * *"

An eminent American veterinarian, in an address delivered some time ago, stressed this very point when he stated:

> It is a fact that all of us are on earth to render service to our fellow man, which we should do with the utmost diligence, but the laws of economics demand that we receive enough in return for our services to permit us a subsistence, as well as a reserve fund for the days when we will not be able to face the hazards of our profession.

Bearing the foregoing points in mind, the following, then, are some of the suggestions and recommendations of your President.

1. Dues: The American Veterinary Medical Association has open before it an immense field in which it may render assistance so as to advance the material and professional interests of its members and for the ultimate good of those served by the profession, but in the past, at least, there has been one insurmountable barrier that has absolutely limited the activities of the national association, and that is, *inadequate funds.* Throughout a year many appeals are received by the general officers, and if some professionally patriotic individual or group of individuals cannot be found to become interested in the matter presented, then the general officers have but one recourse, and that is to state that nothing can be done. In other words, as the immediate past president indicated in his address a year ago, there is undoubted evidence that with the limited funds now available for conducting the business of the Association, the organization is drifting, with no definite constructive objectives. The organization has even been criticized, and in a measure justly so, for accepting from its members the present yearly dues, well knowing that the sum is entirely insufficient for any constructive effort.

At the present time, for example, the American Veterinary Medical Association has such committees as that on Veterinary Biological Products, on Proprietary Pharmaceuticals, on Veterinary Education, and at the Kansas City meeting a Committee on Commercial Slaughter Houses was authorized. In order to function properly, each of these committees should have a full-time secretary to carry on the numerous duties devolving upon them. They are working under tremendous handicaps because of a lack of funds and, furthermore, the members of many committees have full-time, responsible positions, so that they cannot devote to the A. V. M. A. the necessary amount of time for the work to which they are appointed.

Some of the state associations have already taken this matter in hand by increasing their dues and by perfecting an organization which is functioning so efficiently that the state associations having taken this step are being pointed to as models for other state associations to follow. These fortunate states have been able to bring into the animal-health picture not only the veterinary profession, but the financial, live stock, and public health organizations as well.

An inquiry into the fees paid to other national and international professional organizations indicates that the members of the American Veterinary Medical Association are providing a comparatively infinitesimal amount to support the activities of their organization. Many of us are paying much more in the way of dues to fraternal organizations, service clubs, country clubs, etc.—organizations from which we do not hope to profit materially—and we are doing so cheerfully. A similar attitude in regard to the American Veterinary Medical Association would pay large returns in the way of professional and material advancement.

There are those who may say that the present is not a time to advocate increased dues. Your President contends that above all others, the time is opportune to take this step. All American institutions are now undergoing a process of reconstruction, based upon a new concept of financial ability and responsibility. The veterinary profession should also build a firm base at this time—during this reconstructive era—so that it may render the service, and reap the rewards, which the public will demand of it.

With additional financial receipts—a minimum of $8 for the year, to include subscription to the JOURNAL of the American Veterinary Medical Association—there would be available a sum that should permit the initiation of certain very desirable steps

in the work of the American Veterinary Medical Association. Having demonstrated the worthwhileness of these initial steps, and having convinced the membership of this fact, additional steps should then be taken to advance the dues to a maximum of $20 annually. I hope that the Executive Board will give the most serious consideration to this matter and that the members also will urge them to permit the electorate again to express their opinion at an early date.

2. Organization: The necessary funds having been made available, the Association should then turn its attention to the development of and to modernizing its organization so as to make it an effective instrument for spreading the gospel of veterinary medicine.

At no time has the American Veterinary Medical Association —because of financial limitations—and especially during recent years, when the profession has been making a serious attempt to break the fetters that have bound it, been an organization to assist in a national development of a strong program.

The American Veterinary Medical Association needs a competent, full-time business executive or manager, whose chief responsibility would be to pilot the organization over the troubled waters of our professional life and to develop its opportunities. This should be a separate department, distinct from the editorial and journalistic one. Such a business manager should initiate steps for carrying out the national policies of the organization. He should from time to time lay his plans before the Executive Board, and upon their authorization he should at once take steps to make them effective. The organization that we now have has accomplished wonders, but the time has come when we must make positive, constructive efforts to place our organization upon the firm professional basis that it should occupy.

If the immediate future is inopportune for taking this step, I nevertheless hope that when the new House of Representatives is established, it will at once look into the matter of the employment of an executive secretary or business manager.

3. Public and professional relationships: Undoubtedly veterinarians are frequently astonished that their worthy and scientific attainments are not universally acknowledged. Charlatanism is still prevalent to an enormous extent in the handling of animal ailments, and the public is paying a terrific price for it. Of course, this is not a modern evil and is not confined to veterinary medicine, but we are concerned in it particularly as it affects the relationships that should exist between the public

and the veterinarian. It must be said to the credit of the veterinary profession that though it is frequently accused of ulterior motives, it is attempting to do the best that it can to protect the public from its own shortsightedness and faith in empiricism. We see the veterinary profession today doing everything that it can to enlighten the public in regard to the graft that is being practiced upon it by the use of innumerable, valueless live stock remedies. We should go even farther on this problem. It is stated on fair authority that annually in Illinois—and this is doubtless equally correct for other states—owners of live stock probably expend twice as much for nostrums as they pay for veterinary service, exclusive of the cost of anti-hog cholera serum and other biological preparations. *The American Veterinary Medical Association should go on record as being opposed to the direct sale of any remedy that its "Committee on Proprietary Pharmaceuticals" considers of questionable value, or unsuited for use by laymen.*

Possibly one reason for lack of knowledge by the public in regard to veterinary medicine is that our beginning as a profession was a lowly one and that it is one of rather recent origin. It takes a long time to change public thought, and many of those now alive cannot forget the uneducated, unscientific animal health physician of their childhood.

As I see it, there are at least two methods by means of which we may improve the relationship existing between the veterinary profession and the public. *The first of these methods is constantly to improve the character of our work and thus demonstrate our worth, and the second method is by publicity.*

The veterinary profession differs from some of the other learned professions in that it does not have the popular appeal. People are intensely interested in anything that concerns them as individuals, and therefore, their constant search for knowledge regarding medical and dental affairs, but they have only a slight concern in animal-disease problems, and the effect these may have upon the human health, until they are directly affected, beneficially or otherwise, by disease and unprofitable conditions in animal herds and flocks.

Having had a lowly beginning, the veterinary profession is still made the victim of a type of publicity that is very difficult to contend with. During the past year, for example, there has been a motion picture which placed the veterinarian in a very unfavorable light and which was entirely without professional

truth. A foremost American veterinarian, in writing us about this, stated as follows:

> The feature in the picture pertaining to the veterinary profession is distinctly detrimental to the profession, for the reason that it lowers the estimation of the general public for the profession. It portrays the veterinarian on a plane far lower than the oldtime illiterate horse doctor ever was. My reaction to the picture was distinctly unfavorable. It portrays the veterinarian as a petty, political, bulldozing ignoramus.

So thoroughly were we impressed with the harm this motion picture was doing to the profession that we entered into correspondence with the artists' group sponsoring it, but obtained little satisfaction from them. Their position simply was that they had to have a villain, and the veterinarian fitted into their scheme of things. When it was pointed out to them that they might have selected a scenario writer as the villain, they sidestepped the issue. The correspondence in regard to this matter has been placed in the hands of our Committee on Resolutions.

Veterinarians receive unfavorable publicity from other sources. There are certain unethical supply houses dealing in animal biological products that do not hesitate to give the impression in their advertisements that the fact that they are under federal supervision means that the federal government is supporting their claims for the very questionable merit of some of their products. *There might be a remedy for this feature if the federal authorities would withhold from such concerns the advantages of federal inspection if they did not rectify their ways and discontinue using the governmental prestige to further the sale of their questionable products.*

Turning now to the other side of the picture, how may the profession obtain desirable publicity and improve its public relationships? No one denies the truism that if the character of our work justifies it, desirable publicity will be ours. It is equally true that in modern times, no matter how good the character of the services that one furnishes, if one does not keep the public informed of the nature of these desirable services, one's opportunity for doing good is going to be immensely retarded. It is farthest from the thought of your President to recommend direct, blatant or semi-blatant advertising. Physicians and dentists have adopted the policy of doing no direct advertising, and as individuals they adhere to it, and yet they are the beneficiaries of some wonderful campaigns of indirect advertising in their behalf. One need only to read in the daily press the syndicated articles by reputable physicians, writing

on matters of human health, and carefully worded to bring the family physician into the picture, or to hear over the radio day after day, "use a certain preparation twice a day, see your dentist twice a year," to realize how this plan is working out. Even more recently an eastern manufacturer of lenses sponsored a series of intensely interesting articles broadcast over the radio, and delivered by a lecturer in human medicine, which excited far-reaching interest and which indirectly accrued to the benefit of the medical profession.

It is not out of place to mention that during the year the officers of your Association, as well as interested members of the profession, entered into correspondence with the sponsor of the group of lectures referred to in the preceding paragraph, with the objective of having included one or two articles about the veterinary profession. We received very courteous replies, and were assured that some thought would be given to the matter, but that it might be difficult to fit veterinary lectures into the plan as it was already outlined. It was our thought that a series of lectures about veterinary meat inspection, veterinary animal disease control work in tuberculosis, veterinary discoveries in regard to the transmission of insect-borne diseases, and subjects of a similar type could be made of immense interest, and we are still convinced of this, but we made no satisfactory headway with those having charge of this series of broadcasts.

As another example, we may indicate that during the year a large eastern life insurance company carried a series of full-page advertisements lauding the medical profession for its valuable service in controlling human tuberculosis. The veterinarian, in his great fundamental work in stamping out animal tuberculosis, and thus reducing immensely the human infection, was entirely ignored. A letter directed to the president of the insurance company brought a very courteous reply, in which the writer stated that he was aware of the services of the veterinarian in the control work in regard to tuberculosis, but the veterinarian still lacked popular appeal, and their advertisements were written largely with the primary objective of getting the public to read them. We were informed that if the situation developed properly, the veterinary profession would receive the same consideration in future advertisements by the insurance company in question.

I have cited the foregoing instances merely to show that the indirect publicity which the medical and dental professions is receiving gratuitously is, after all, basically owing to the fact

that the sponsors of this form of publicity feel that it is going to be of specific benefit to the firm, individual, or group of individuals paying for the advertising. Until the time arrives that the veterinary profession can fit into this picture, we cannot hope for the type of publicity that has been described in the preceding paragraphs.

The picture is not so dark, however, as it might appear upon the surface. Some of our veterinary supply houses have taken the matter into their own hands, and their display advertisements in live stock and other journals have undoubtedly created a favorable professional atmosphere for hundreds of practitioners of veterinary medicine.

There still remain many openings or avenues by means of which the veterinary profession may place itself in a favorable light before the general public.

(a) If in its service to the public press—and relating to the activities of the veterinarians in its employ—the U. S. Department of Agriculture would designate these employés as *"veterinary inspectors,"* instead of Department of Agriculture or Bureau of Animal Industry inspectors, it would be giving more accuracy and specificity to its reports to the public, *and the profession would receive deserved credit and publicity.* Because, for example, press reports state that an outbreak of foot-and-mouth disease is being stamped out by "U. S. Department of Agriculture inspectors," or that the federal meat inspection service is being performed by "U. S. Department of Agriculture inspectors"—and many other instances may be cited—the public is totally unaware of the services of the veterinarian in this connection. In fact, agriculture receives full credit for a service performed by the veterinarian. *Your President recommends that this matter be officially called to the attention of the Secretary of Agriculture and that an attempt be made to rectify what is doubtless an unintentional injustice.*

(b) Those veterinarians occupying public positions possibly have greater opportunities for creating a favorable impression for the profession than those not so situated. Officers of our veterinary schools have it in their hands to so guide the professional lives of the young men entrusted to their care that the latter will carry with them from the halls of learning a professional attitude and bearing which must inevitably redound to the profession as a whole. Also, veterinarians in public positions should take every opportunity to

appear in a professional capacity before the public. In many of our educational institutions there are broadcasting stations, and the directors of these stations welcome educational talks by veterinarians about their work and activities. It is needless to state that addresses of this nature must be carefully worded, or they may do more harm than good.

(c) Veterinarians in private practice, as well as those publicly employed, should not miss an opportunity to present their profession in the various service clubs, to appear on the programs of these service clubs and to address the members on timely subjects of veterinary interest.

(d) There are many organizations of women's clubs that are intensely desirous of increasing their knowledge about meat and milk inspection and the sanitary production of these human foods. Veterinarians can do their profession no greater good than to exert themselves in every possible manner to spread this gospel.

(e) In certain states of the Union, veterinarians either as individuals or as officers of state associations have been able to obtain a very high grade of publicity for the profession by the publication of carefully worded articles in the agricultural and lay press. The writer has had an opportunity to judge the value of these articles, and has been impressed by their immense worth.

(f) Recently our attention was called, by the editor of one of our leading professional journals, regarding another possible source of intraprofessional publicity. The suggestion was that the national organization, the state organizations, or possibly our veterinary schools, should at once proceed to obtain phonographic records in which leaders of the veterinary profession would leave an indelible record, in their own words, of some of their activities which, in a measure, have been instrumental in placing the profession in its present more or less enviable, though little known, position. If we today had phonographic records by Law, Salmon, Dalrymple, and a large host of others who have passed to the great beyond, how invaluable these would be for the education of our embryonic veterinarians. We should no longer delay obtaining such records from those who are still with us.

The subject of public professional relationships is such a very fruitful one that its discussion could be continued almost indefinitely. It is hoped that enough of it has been pointed out and

that the funds will be furnished and the organization perfected so that the veterinary profession of America may take full advantage of its opportunities, and that the public may derive a due measure of veterinary service by having its interest in this profession stimulated.

III. Some A. V. M. A. and Professional Opportunities

1. Medical relationships: Veterinary medicine is in every sense of the term a medical science and, for this reason, if for no other, the American Veterinary Medical Association should seek very close affiliation with other organized medical groups, especially the American Medical Association. The two associations have much in common, they need each other's help, and there should be an interchange of ideas. The support of both organizations is essential for questions of human health insofar as they are influenced by animal diseases and animal food-products. From the strictly scholastic standpoint, the veterinary schools of America may well pattern after the medical schools. It is urged that every reasonable effort be made to enter into closer relationship with the American Medical Association.

2. Agricultural relationships: Neither must the American Veterinary Medical Association forget that agriculture is its foster mother, and anything that tends toward insulation against agriculture is certainly to be condemned. From the material standpoint, agriculture can do more for veterinary medicine than any other group. Similarly, veterinary medicine has demonstrated in the past, by its numerous controls over widespread outbreaks of animal diseases, that it has more than repaid agriculture for the early fostering care received from the latter. There are still many ways in which veterinary medicine can be of service to agriculture in addition to what must always be its major activity—the control and relief of animal diseases.

The individual members of the veterinary profession probably have a closer contact with agriculturists than any other group of professional men. This very close relationship is the result of years of mutual endeavor and coöperative help. It is built upon the firmest of human relations, those of friendship and respect.

As an indication of how the veterinarian may be of additional service, it may be mentioned that in many sections of the country there is still a woeful lack of the better classes of live stock. A bulletin from one of our northern middle western states, for example, states that the steers marketed from that state were

almost equally divided between the three top grades and the three low grades on the market—one-half of the cattle classified as common, cutter, or low cutter. In this same state there has been a most insistent demand for farm relief. Had the common steers been sired by a good bull, they would have sold, on their merit for beef, 20 per cent higher. They would doubtless have weighed 100 pounds more each, and could have been marketed at an earlier age.

Animal breeding experiments have demonstrated that the use of a good bull added at least $11 to the value of each of the calves produced from good cows, and yet purebred bulls at the present time are a drug on the market. A well-informed writer on agricultural economics recently stated that in his opinion, one of the greatest sources of farm relief is available in the purchase of good sires.

The foregoing is cited simply to indicate that veterinarians, in their close personal relationships with agriculturists, can exert an immense amount of good, to encourage the purchase not only of better beef bulls, but better sires of all species of live stock. Indirectly, this will again redound to the material welfare of the veterinarian.

3. The extension veterinarian and his functions: A moot question in many sections of the United States centers around the character of service rendered by so-called extension veterinarians. As a rule, the general policy governing the activities of these public servants is quite well defined. I believe that in most states the policy of the extension service in regard to animal disease is that the work of the extension veterinarian should be almost purely educational, enlightening the owner of live stock on what he can and should do for himself, particularly in practicing the principles of sanitation, the giving of his full coöperation in the enforcement of regulatory measures, encouraging the live stock owner to use the services of a competent graduate veterinarian when needed, and always bearing in mind that it is not a good procedure for the extension veterinarian to do the work of the regular local practitioner of veterinary medicine. This policy is good insofar as it goes, but it relates almost entirely to the education of the layman and while no one denies that enlightenment about animal-disease questions is needed in many sections, still there is an even greater need for the services of an extension veterinarian whose almost exclusive duty it shall be to carry, to practitioners of veterinary medicine in the field, the results of the labors of the research worker and

of the teacher, and, on the other hand, carry from the practitioner back to the classroom and laboratory, the practical results obtained by the workers in the field. If such a type of veterinary extension service can be developed, it is going to result in a better qualified veterinary practitioner, and as the practitioner is the fountain-head of veterinary knowledge insofar as agriculture is concerned, therefore it must redound to the great benefit of agriculture and other interests employing veterinarians.

Other services that the extension veterinarian may well take upon himself—and it is admitted that indirectly the veterinarian will profit from these services, but directly the greater benefit is to those served by veterinary medicine—is serving as an intermediary between the agriculturist and the veterinarian in spreading information about the immense benefits for agriculture in the development of plans for animal-parasite control, abortion-disease control, and similar problems. In many sections of the United States, when the live stock interests have once had demonstrated to them the improved health conditions of their live stock by a limited application of the principles of parasite control, the idea has at once spread, so that in many states there are several counties in which live stock is virtually free from some of the most harmful parasites. During the year, it has been the privilege of your President to establish direct contact with some of the communities in which a veterinary service of this nature has been rendered, and we found only expressions of general satisfaction.

What has been said about parasite control may almost be repeated word for word about those communities and farms in which a sincere and intelligent effort has been made to stamp out abortion disease. It is well here to give a word of warning, as there is a serious threat that those not fully familiar with the fundamental principles of animal disease control and with serological and other biological tests, will, in their enthusiasm to obtain immediate results, seek to do work of this nature themselves, or delegate it to those not qualified. A somewhat comparable situation, and in a measure a deplorable one, exists in various sections of the United States in regard to the blood test for the detection of pullorum disease in poultry. Unfortunately this latter work has fallen, to a considerable extent, into the hands of the improperly qualified, the unscrupulous, and those having a personal interest in the results of the tests, so that in many regions this test—a thoroughly worthwhile one—

is virtually discredited. Veterinarians must be constantly on their guard to see to it that the abortion blood-test and other animal serological and laboratory tests do not fall into equal disrepute. It is only by the closest coöperation of extension veterinarians, practicing veterinarians, laboratory workers and state sanitary officials that this entire matter may be safely guarded for the best interests of those whom we seek to serve.

4. Poultry and sheep practice: It is not amiss at this time to mention another veterinary activity that is going to slip away from practitioners unless they demonstrate their value in the handling of poultry and sheep diseases. During the past year, it has been forcibly brought to our attention that, in some sections of the country, poultry disease work is getting away from veterinarians because many poultry-owners have not had demonstrated to them the veterinarian's ability in this connection, and also because many veterinarians refuse to take an interest in poultry practice. Our veterinary schools and our veterinary journals, as well as the American Veterinary Medical Association, may well take an interest in this matter so as to stimulate veterinary thought along proper channels in this connection.

5. Veterinarians as meat, milk and sanitary inspectors: There is probably no greater question today before the veterinary profession as a whole than that of the development of meat, milk and sanitary inspection service for many American communities that are now without it. Many people have but little knowledge of animal diseases which alter the character of flesh and make it objectionable and possibly harmful. The public undoubtedly takes it for granted that some national, state or municipal body is aware of the situation, and will do for the people those things which they as individuals are unable to do for themselves in matters of health. This is a proper conception of government, and the various states and municipalities should provide for the inspection of their food products.

Our national meat inspection law—a model for the rest of the world—does not permit the inspection of animal food products when these products do not enter interstate commerce. The federal service has been extended so that the regulations require that establishments to which inspection has been granted shall operate their entire plants under federal meat inspection, even though only a relatively small part of the output may actually be transported interstate. Nevertheless, *it is estimated that at present the federal government is able under existing law to*

apply this inspection to approximately only two-thirds of the total amount of meat prepared and largely consumed in the United States. This leaves approximately one-third of the domestic meat supply without adequate inspection. An eminent authority states that the loathsome and dangerous conditions found in the two-thirds of the animals slaughtered for meat certainly show the necessity for extending inspection to the one-third.

During the fiscal year ending June 30, 1931, there were slaughtered and veterinary-inspected in the 334 federally controlled abattoirs approximately seventy-four and one-half millions of animals. This, then, means that there were probably slaughtered and sold in addition, in the United States, as meat food products, at least one-half of the above-mentioned enormous numbers of animals, without veterinary inspection. It is interesting to note that the total of meat and meat food-products condemned last year in federally veterinary inspected slaughterhouses was estimated to be sufficient to supply more than half a million people with meat at the present yearly per capita consumption.

Certain evils have grown up because of this partial application of the principles of meat inspection to two-thirds of the animals slaughtered in the United States and not to the remaining one-third. In federal veterinary meat inspection there is, of course, antemortem inspection. It has been stated that animals failing to pass an antemortem inspection are promptly shipped from the control of federally inspected plants and are slaughtered in non-inspected plants where the product may enter state traffic without impediment or control. Live stock producers and breeders have learned from experience that it is more profitable to sell their diseased and suspicious animals to local country butchers and establishments operating without official inspection, than to ship them to public markets where they will be subject to federal veterinary inspection.

Furthermore, when there is no efficient system of inspection, the sanitary condition of the slaughter-houses is many times in a deplorably filthy condition.

Those states not having an efficient system of state meat inspection, probably do not realize the handicap under which they have placed their own live stock industry. If it happens that these states do not have located within their borders a slaughtering plant where federal inspection is maintained, then animals must be shipped to another state in order to obtain the advan-

tages of federal inspection, to be followed by a return of the federally-inspected meats to the state of origin for human consumption. This back-and-forth shipping of animals and animal food-products is a terrifically expensive one to the people of those communities subjected to it, and would be largely obviated by efficient systems of state veterinary meat inspection.

If the public could be made to realize fully the benefits of veterinary meat inspection, our entire meat supply would now be receiving either federal, state or municipal inspection. In some states, notably Pennsylvania and California, because the people were aroused, systems of state meat inspection have been established which are to all intents and purposes as efficient as federal inspection. Forty-two cities in the United States have ordinances requiring inspection of that part of the local meat supply which is not governed by veterinary inspection. This is a step in the right direction, but 42, among approximately 2,700 incorporated cities and towns in the United States having a population of 3,000 or more people, is far from meeting the present needs.

Amongst the larger cities establishing meat inspection within the last year, particular mention should be made of Saint Louis, Missouri. The first annual report, covering six months of municipal veterinary meat inspection in Saint Louis, has just been issued. In that city, upon the inauguration of meat inspection, there were found a group of slaughter-houses that, during all the years of federal inspection, had been operated without supervision of any kind. Nevertheless, by tactful handling on the part of the municipal veterinary inspectors in Saint Louis, these slaughter-houses have been brought within the provisions of the very excellent meat inspection law operative in Saint Louis. Those plants that had been without inspection have been transformed into sanitary meat establishments, *and in some instances far exceeding the actual requirements, so that these plants now are veritable meat laboratories.* This new service has not disrupted, to any degree whatsoever, the slaughter of live stock and the processing of animal food-products in Saint Louis, *and it has given the people of that community a protection against transmissible animal diseases and against the consumption of diseased, loathsome food-products that could have been obtained in no other manner.* More than 50 per cent of the meat consumed in the City of Saint Louis and Saint Louis County is prepared in these municipally veterinary-inspected plants. *The*

City of Saint Louis may well feel proud of the standard it has set for other American cities.

Not only would state systems of veterinary meat inspection provide protection against the transmission of animal diseases, but meat inspection statistics made available would be of great benefit to the live stock industry. For example, much additional information is needed on the large subject of the distribution and prevalence of parasites in the various states. A system of state meat inspection would provide the facilities for obtaining this information of great value to live stock producers.

Again, a system of state meat inspection would provide safeguards against frauds that may now be practiced in the preparation of animal food-products. The addition of large percentages of tallow to lard, the mixing of high percentages of cereals with sausage meat, the addition of large quantities of water, the use of artificial coloring matter so as to disguise the questionable products, and selling such products in competition with those produced in inspected plants, are not only harmful to the consumer, but to the producer as well.

Why is it that the veterinary profession has failed to realize the opportunity to assist in the protection, not only of public health, but also of the live stock industry, by supplying it with information made available by the federal meat inspection service? It seems that in many instances the veterinary profession has considered it the duty of boards of health to take the initiative in veterinary meat inspection matters, on the ground that it is primarily a public-health measure. Your President believes that this is a mistake. Every veterinarian in the United States should take it upon himself, by carefully worded public addresses, and utterances through the press, to call attention to the present situation in regard to the meat food supply of the people of the United States. Veterinarians alone have this information, and failing to make the public cognizant of these affairs is not only failing to do our duty, but is also failing to grasp an opportunity.

The American Veterinary Medical Association, the veterinary journals, and the veterinary supply houses in the United States should assist in putting on a campaign to popularize this subject to our profession. If finances were made available and the proper officers provided, such a campaign should be organized by the American Veterinary Medical Association, it should be the central office, and it should compile information on all the phases of this subject so that it might be available for those

communities in which an interest had been aroused on the question of proper food inspection. Such information might include ordinances suitable for cities of different sizes. At the same time, the veterinary journals should be encouraged to publish a series of articles covering the various phases of meat and milk inspection adequately. It is sincerely urged that this matter be given the most serious attention of all those in the veterinary profession.

6. *Organize veterinary groups:* The American Veterinary Medical Association should at once take steps to encourage the formation of small groups of veterinarians in contiguous territory—the county unit will in most instances be found to be too small—to have monthly or quarterly meetings, in afternoon or evening session, for the interchange of professional experiences and thought. Many of these groups are already functioning efficiently in various parts of the United States, and so great has been the mutual good the members have derived from these gatherings that it is a serious mistake for the A. V. M. A. to fail to take a most aggressive attitude in the formation of many more of these local professional groups. Never in the history of organized veterinary medicine has 100 per cent affiliation of graduate veterinarians with their societies been so essential as at this time.

7. *Practice acts and reciprocity:* The American Veterinary Medical Association has before it another large field in directing education regarding our various state veterinary practice acts, and reciprocity between the states insofar as the licensing of veterinarians is concerned. It is conceded that, to a considerable extent, local state conditions govern the practice acts of those states, and also whether the state in question shall enter into reciprocal relations with the neighboring states or with those in more distant parts of the Union insofar as recognition of its veterinary license is concerned. The American Veterinary Medical Association, in spite of the foregoing, might well prepare model state veterinary practice acts, and, by frequent articles upon the subject, encourage the recognition of a license from one state by others.

8. *Veterinary surveys:* The veterinary schools of North America are at this time graduating approximately 200 veterinarians each year. Your President is fully convinced that 50 per cent of these young veterinarians are hopelessly muddled as to how best to take advantage of their recently acquired professional education. Many of them are desirous of rendering the

greater good by entering the field of private veterinary practice, but there is no source of information available to guide them in their choice of a location. The American Veterinary Medical Association should take the lead in furnishing data as to the distribution of the veterinary population, the live stock population, the need of veterinary services such as meat and milk inspection, the federal Department of Commerce data regarding the financial standings of the various communities, and whether the communities are engaged in live stock production or whether they are primarily industrial or largely agronomic. Information of this character, kept up to date and made available not only to recent graduates, but to improperly located practitioners of veterinary medicine, would be of inestimable benefit.

9. Veterinarians and boards of health: Boards of health, in a large number of communities, do not have a veterinarian as a part of their membership. Anyone having a knowledge of the interrelationship existing between human health and animal health, and knowing the numerous intricate professional problems that must be solved in this situation, must fail to understand why boards of health are organized without taking advantage af the services of a veterinarian. Education in the veterinary ranks and amongst boards of health and in the public press, is essential to obtain what should not necessarily be considered as a recognition of the veterinary profession, but for the public good. The American people are not receiving the benefits of the knowledge that has been developed in the veterinary profession, and they are entitled to it. It is a duty of organized veterinary medicine to see to it that they get the necessary information.

IV. Group Insurance

Your President feels that he cannot close this address without making a recommendation in regard to the advisability of obtaining group insurance for the membership of the A. V. M. A.

In such a group, of which I have a personal knowledge, the average premium is $13.32 the year for each $1,000 of insurance, and the dividend on this in the absence of death losses has varied from forty to sixty per cent, so that the cost per year for each $1,000 of insurance has been from $5.33 to $7.99. This rate is so ridiculously low and the benefits are so incomparably high that our Association can not forego the opportunity of making a thorough investigation of this entire matter. This form of insurance may be had without a medical examination—provided

it is taken by the members within a reasonable time after it becomes operative, or within a reasonable time after an applicant becomes a member of the Association. If taken it will add very materially to our estates, and is for the protection of our dependents. I recommend that a special committee be appointed to investigate this matter and report back to the Association at its next annual meeting.

V. Summary

1. Increase the annual dues, from a minimum of $8 to a maximum of $20 the year.

2. Reorganize, the reorganization to include the plan presented by the Special Committee on Affiliation of State and Provincial Associations with the American Veterinary Medical Association, and also to include the appointment of an executive secretary or business manager.

3. Public and professional relationship to be improved by means of approved and ethical publicity in order to educate the public regarding our scientific and professional attainments, and our ability to render service for their best interests.

4. Attention is called to some A. V. M. A. and professional opportunities such as medical and agricultural relationships; the extension veterinarian and his functions—including parasite and disease control; poultry and sheep practice; meat, milk and sanitary inspection; encourage the organization of local societies of veterinary medicine; formulate veterinary practice acts, and professional interstate reciprocity relationships; prepare a veterinary survey; and veterinarians and boards of health. *Our members should be assisted to the fullest extent of our united and collective ability.*

5. It is recommended that an investigation be made of the possibilities of "group insurance" for our members.

VI. Conclusions

In the preparation of this address, your President wishes to acknowledge his entire indebtedness for sources of information to our veterinary journals, to veterinary professional addresses, copies of which have freely and unselfishly been supplied him, and to the many individuals in the profession whom he has been privileged to contact throughout the year. In many cases these various sources have been quoted literally and extensively. Your President has felt it to be one of his duties to attempt, insofar as it is possible, to concentrate in this address the veterinary thought of the members. If he has done this in a small

measure, and if, furthermore, he has aided, by his participation in professional programs, in cementing the veterinary conduct of our widespread domain into more or less common and mutually beneficial channels, he feels that he has done his mite for the profession that holds our life interest.

When he mentally reviews the immense possibilities in the field of veterinary endeavor—how much has been done, and still how little when compared with that undone—he feels appalled. But there always arises the American Veterinary Medical Association, which can, if given the opportunity, exert a terrific influence for good.

No one who has had the opportunity to observe veterinarians in all parts of the United States flocking to local, state and sectional veterinary meetings, can ever accuse this profession of smugness. Nevertheless, we also need to develop a courageous leadership. There must, within reasonable limits, be a unanimity of professional thought and conduct. We must establish a program of public education in every section of our territory. We should retain within our ranks only those who are rendering the highest type of approved service, men honest with the public and with themselves, and we should strengthen our morale by a faith in veterinary medicine based upon its high accomplishments. And finally, it will do us all good to renew our allegiance to that unofficial code of ethics formulated some time ago by an unknown writer:

To regard my profession as something more than a means of livelihood;
To value character more highly than reputation and truth above popularity;
To be merciful and humane, preventing needless suffering among dumb beasts;
To be faithful and zealous, preventing needless loss to those I am called on to serve;
To guide my conduct by sober judgment and my judgment be a never-sleeping conscience;
To be modest and open-minded and thankful for every opportunity to increase my knowledge and my usefulness;
To be a co-worker with my fellow practitioners by the mutual interchange of counsel and assistance;
To be true to myself, measuring my success by the value of the service I render rather than by the fee I receive.

Louisiana had just half a dozen veterinarians at Atlanta: Drs. H. H. Baur and L. H. Bennett, Monroe; F. B. Ford and Hamlet Moore, New Orleans; A. D. Kendrick, Homer; Harry Morris, Baton Rouge.

RESULTS WITH PIGEON-POX VIRUS FOR THE IMMUNIZATION OF FOWLS AGAINST CHICKEN-POX*

By S. T. MICHAEL, *Berkeley, Calif.,*
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Doyle and Minett,[1] in 1927, pointed out the considerable resistance of pigeons to fowl-pox and the susceptibility of chickens to pigeon-pox virus. They claimed that while the viruses of chicken-pox and pigeon-pox are immunologically indistinguishable, they differ in their species adaptation. They apparently succeeded also in immunizing chickens against chicken-pox with pigeon-pox virus.

Zwick, Seifried and Schaaf,[2] in 1928, found that fowls vaccinated with pigeon-pox vaccine, consisting of a suspension of powdered pigeon-pox scabs in 50 per cent glycerin-saline mixture, showed strong resistance to artificial infection with chicken-pox virus and an absolute immunity to natural infection in three weeks following vaccination. From studies in the field they concluded that "the vaccination gives best results when used to suppress outbreaks on infected farms."

Doyle,[3] in 1930, reported on the use of a vaccine consisting of 1 per cent suspension of powdered pigeon-pox virus in a mixture of 80 parts of glycerin with 20 parts of physiological saline. He states that the vaccine confers considerable, but not complete, protection against severe artificial infection with chicken-pox virus and that solid immunity against natural infection is established about the fourteenth day after vaccination. This vaccine, according to Doyle, does not give rise to any constitutional disturbance or loss of condition, nor does it interfere with egg production.

Johnson,[4] in 1931, concluded from the use of pigeon-pox vaccine, prepared according to the method of Doyle,[3] that this product is a very satisfactory immunizing agent for preventing natural infection of chicken-pox, but is not 100 per cent efficient in immunizing against artificial infection. The same writer,[5] in 1932, reported that pigeon-pox vaccine conferred protection against disease under natural conditions for at least one year.

PREPARATION OF PIGEON-POX VACCINE

For the experiments reported herein, pigeon-pox virus was obtained by permission of the U. S. Bureau of Animal Industry,

*Presented in part at the annual meeting of the California State Veterinary Medical Association, Pasadena, Calif., June 15-18, 1931.

through the courtesy of T. M. Doyle, of the Ministry of Agriculture, England. This virus was only mildly virulent for pigeons when received, but, after four or five passages, it became capable of producing marked lesions at the point of inoculation in susceptible pigeons.

The technic recommended by Beach,[6,7] in 1927 and 1929, for preparing chicken-pox vaccine was followed. A suspension of pigeon-pox virus was applied to the skin of the breast and legs of pigeons from which feathers had been plucked. On the tenth to the fourteenth day, depending on the rapidity of development of lesions, the pigeons were sacrificed. The lesion tissue was removed by excising the skin, passed through a Latapie grinder twice and then through a 60-mesh sieve, with the addition of a sufficient mixture of equal parts of glycerin and 1 per cent phenolized saline to give a suspension of 0.25 grams of virus (lesion tissue) in each cubic centimeter. The concentrated vaccine was stored in the refrigerator. Just prior to administration, it was diluted sufficiently to make each cubic centimeter contain 0.002 gram of lesion tissue.

Some of the lesion tissue produced as described was desiccated in a vacuum jar with phosphorus pentoxid for preparation of vaccine with desiccated virus according to Doyle's[3] method.

Tests for Virulence of Pigeon-pox Virus for Fowls

A 1 per cent suspension of powdered dry virus in physiological saline was applied to a scarified area on the combs of six cockerels. Lesions, consisting of minute white eruptions, appeared on the eighth day. They healed within fourteen days without spread or scab formation.

In another group of twelve cockerels, six were inoculated by the application of a vaccine, prepared by Doyle's[3] method from desiccated virus, to the skin and feather-follicles on the leg from which the feathers had been plucked. The six other cockerels were treated in the same manner with vaccine prepared from fresh lesion tissue. The exposed follicles on all birds were swollen and reddened on the seventh day, later became yellow in color and resembled in appearance a young chicken-pox lesion (fig. 1), after which they became cornified and covered with dry scabs. The surrounding skin did not become involved and healing was complete in from sixteen to twenty days. There was no discernible difference in the type of "takes" or in the effect on the general health of the birds produced by the two types of

vaccine. Like results were obtained by inoculations of additional chickens.

The Susceptibility of Fowls Vaccinated with Pigeon-pox Virus to Artificial Infection with Chicken-pox Virus

For these tests, cockerels were vaccinated with pigeon-pox virus by the follicle-infection method and subsequently exposed to virulent chicken-pox virus, either by direct inoculation or by severe contact exposure. Only those fowls on which definite involvement of the follicles had occurred were used.

In the first tests, virulent chicken-pox virus was applied to a scarified area on the comb of each of sixteen vaccinated chickens

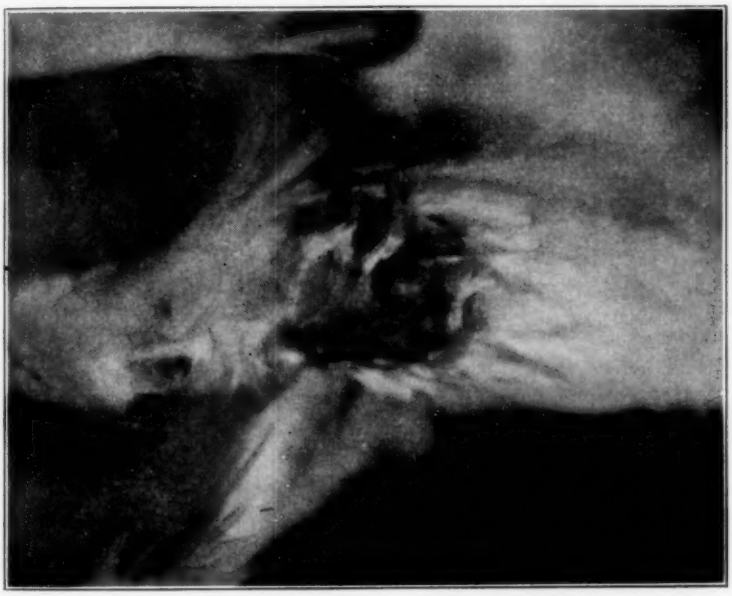

Fig. 1. "Take" from pigeon-pox vaccine, on the eighth day following vaccination.

and eight controls. The follicles of the vaccinated cockerels had entirely healed from two to four weeks previously. Marked lesions of chicken-pox developed on all of the vaccinated and control fowls.

In the second test, six cockerels that had been vaccinated thirty days previously and six controls were placed in a small pen with four cockerels actively infected with chicken-pox. They were kept under observation for a month, during which time two of the vaccinated fowls and one of the controls contracted a mild form of chicken-pox.

In the third test, three birds that had been vaccinated by the follicle method three weeks previously were placed in a cage that had been repeatedly used for chickens infected with chicken-pox without intervening disinfection. The combs of the fowls were lightly scarified to provide an avenue of entrance for the infection. They developed extensive lesions of chicken-pox within a month.

These results fail to show that the vaccination of fowls with pigeon-pox vaccine increases resistance to artificial infection with virulent chicken-pox virus.

Tests of the Susceptibility to Natural Infection with Chicken-pox of Fowls Vaccinated with Pigeon-pox Virus

In these tests, fowls known to have been free from chicken-pox but on farms on which chicken-pox had previously occurred were vaccinated by the follicle-infection method with pigeon-pox vaccine prepared from fresh lesion tissue or from desiccated virus from a commercial source.

The vaccinated fowls were examined individually at appropriate intervals to determine the nature of follicle reaction following application of the vaccine and the numbers that later became infected with chicken-pox. Observations were made on the effect of vaccination on the general health of the fowls and on egg production.

A total of 9,805 fowls, varying in age from three months to two years, on thirteen farms, became available for these field trials during the fall and winter, 1930-31.

In six flocks, comprising 4,611, pigeon-pox vaccine only was employed and all fowls were treated. Vaccine from a commercial source* was used for two of these flocks.

In two flocks (4 and 6) 1,838 fowls were vaccinated and 170 left unvaccinated for controls.

In seven flocks, 3,375 fowls were treated with pigeon-pox vaccine and 1,819 with chicken-pox vaccine. In the latter case the fowls treated with different types of vaccine were housed separately.

In all flocks, lesions in the follicles of the type previously described were detected on practically all of the fowls. The general health of the fowls and egg production were not influenced in any way. Data concerning the later occurrence of natural infection with chicken-pox among them is given in table I.

*The writer is indebted to Dr. W. E. Brandner, of Petaluma, for the information on the use of commercial vaccine.

TABLE I—*Occurrence of natural infection with chicken-pox in vaccinated fowls.*

FLOCK	AGE OF FOWLS (MONTHS)	KIND OF VACCINE	FOWLS VACCINATED	OCCURRENCE OF CHICKEN-POX	
				FOWLS AFFECTED	TIME AFTER VACCINATION (DAYS)
1	4	P. P. C. P.	55 51	0 0	
2	9	P. P.	238	42	61
3	5	P. P. C. P.	359 250	18 0	65
4*	8–18	P. P. C. P.	950 198	23 0	30
5	4	P. P. C. P.	264 195	25 0	300
6†	9	P. P. C. P.	888 317	227 0	30–65
7	6	P. P.	264	0	
8	3½	P. P. C. P.	410 393	0 0	
9	12	P. P.	709	300	37
10	4	P. P. C. P.	449 415	0 0	
11	9	P. P.	600	450	60
12‡	10	P. P.	600	300	45
13‡	9	P. P.	2200	1100	30

C.P. = chicken-pox. P.P. = pigeon-pox.

*70 unvaccinated controls were left in this flock. In one pen, 18 were vaccinated with pigeon-pox and 18 left unvaccinated. In this pen, only 2 fowls of the control group developed chicken-pox and none in the vaccinated lot or the remaining controls.

†In one pen, 72 were vaccinated with pigeon-pox vaccine and 35 used as controls. Chicken-pox occurred in this pen, affecting 32 fowls in the vaccinated group and 19 in the control. 65 unvaccinated controls were left in another pen and fowls in the adjoining pens were vaccinated, one pen with pigeon-pox vaccine, the other with chicken-pox vaccine. Two months later chicken-pox infection occurred in the pigeon-pox vaccinated pen, affecting 19 out of 86 birds. No cases were observed in the control or chicken-pox vaccinated pens.

‡Vaccine from commercial source.

It will be seen from the table that natural infection with chicken-pox occurred among fowls vaccinated with pigeon-pox vaccine in nine of the thirteen flocks in from one to ten months

following vaccination. The number of fowls to become infected varied from 2 to 75 per cent and in one flock (11), in which diphtheritic lesions predominated, there was severe mortality. None of the fowls that were vaccinated with chicken-pox vaccine have become infected.

In flocks in which natural infection with chicken-pox was not extensive, the affected pullets were promptly isolated, thus decreasing the opportunity of contact infection. In flock 6 the lesions that occurred among the fowls vaccinated with pigeon-pox vaccine and the unvaccinated controls were of equal severity.

Natural infection with chicken-pox has not occurred in four of the flocks of young pullets during the interval (one year) since vaccination. As there were no controls in these flocks, it is not known that their freedom from chicken-pox is due to the protection afforded by vaccination.

SUMMARY

Pigeon-pox virus, in the form of fresh lesion tissue and desiccated scabs suspended in glycerin and physiological salt solution, was used for the vaccination of fowls by the "feather follicle" infection method. Reactions resembling mild chicken-pox lesions were produced on the combs and in the follicles. Increased resistance of fowls vaccinated by the "feather follicle" infection method to artificial or natural exposure to chicken-pox virus, however, could not be demonstrated. These results are not in agreement with those of Doyle,[3] Zwick, Seifried and Schaaf[2] previously mentioned, nor with those that have been reported by Johnson[4,5] since these experiments were completed.

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[5]Johnson, E. P.: Pigeon-pox virus and immunization against chicken-pox in fowls. No. Amer. Vet., xiii (1932), pp. 47-48.

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Three Kentucky veterinarians enjoyed the meeting: Drs. John Baird, Lexington; Robert F. Fisher, Paducah; Harry Gieskemeyer, Fort Thomas.

INFECTION OF PIGS AND OTHER ANIMALS WITH KIDNEY WORMS, STEPHANURUS DENTATUS, FOLLOWING INGESTION OF LARVAE*

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INTRODUCTION

In an earlier paper the writers[5] called attention to the fact that in experiments with kidney worms, *Stephanurus dentatus*, using pigs which were litter mates, the host animals became infected with the parasites when the infective larvae were experimentally administered by mouth, but escaped infestation when the infective larvae were placed on the intact abdominal skin. When the skin was scarified, however, so that only the superficial layers were broken, the larvae penetrated readily and worms were later recovered from various thoracic and abdominal organs; gradually most of them reached the renal region and remained there. Similar results were obtained when the infective larvae were injected subcutaneously. The writers' observations were in disagreement with those of Bernard and Bauche,[1] who concluded, on the basis of experiments carried out by them in Annam, French Indo-China, that the specific lesions produced by kidney worms depended upon the path of entrance of the larvae into the body of swine, the oral route resulting in the production of liver lesions and the percutaneous route resulting in the production of renal lesions, including perirenal and periureteral cysts in which the adult worms were contained. The writers have already shown[5] that in experimental percutaneous infections, the larvae invaded the liver with resultant production of lesions. It was shown also that the larvae appeared in the liver, and less regularly in the lungs and other organs, long before the renal area became infested. The data obtained by the writers pointed to a direct migration of the incompletely developed worms from the liver and possibly from other abdominal organs to the perirenal fat, in which location the worms encysted and from which they established channels to the ureters which they ultimately perforated. This afforded an outlet for the eggs which could be discharged to the outside with the urine, thus completing the life cycle. The object of

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the present paper is to present and discuss the protocols dealing with the course of experimental infections of pigs and other animals to which the larvae were administered by mouth.

Experimental Infections Through the Oral Route

Bernard and Bauche[1] reported that they administered infective larvae of *Stephanurus dentatus* to six pigs by mouth. Two pigs, killed one month after experimental feeding, showed hepatic lesions at necropsy but the kidneys and ureters showed no evidence of infestation; the remaining four pigs, killed from 90 to 100 days after experimental feeding of larvae, showed characteristic liver lesions at necropsy but no lesions in the kidneys and ureters. Their conclusions regarding the failure of *Stephanurus* to reach the renal organs following experimental feeding appear to be based entirely on the above experiment.

The writers' investigations on infections through the oral route were begun early in 1927 and carried out from time to time, as favorable opportunities arose, until and including 1930. The technic of culturing and isolating the larvae has already been discussed in a previous paper.[5] The pigs used in these experiments, obtained from a stock free from kidney worms, remained with the sows until they were from 6 to 8 weeks old, at which time they were weaned. In all cases the pigs used in experiments described in this paper were farrowed, and kept subsequently under sanitary conditions in pens having concrete floors, until the termination of each experiment. All experiments described in subsequent pages were controlled, either by keeping some of the litter mates of the pigs involved in the experiments as check animals, or by keeping pigs from other litters as controls, under conditions identical with those under which the experimentally infected pigs were kept. In addition, a number of pigs, involved in various other experiments, were kept under observation prior to, during and subsequent to the time that the kidney-worm experiments were in progress. In no instance did any of the control pigs, kept in pens having concrete floors and with due attention to the cleanliness of the quarters, develop an extraneous infestation with *Stephanurus dentatus*, as determined by careful postmortem examination.

Experimental Infection of Pigs

Experiment I: On February 24 and 28, and on March 2, 1927, each of two pigs (17 and 18), about 2 months old, was given rich cultures of infective kidney-worm larvae by mouth.

Pig 17 was killed on April 20, 48 days after the last feeding

with larvae. The following lesions and worm infestations were noted at necropsy:

Surface of liver roughened, lighter in color than normal, and showing evidence of a more or less recent peritonitis; interlobular connective tissue apparently increased; a number of yellowish nodules, from 0.5 to 1 cm. in diameter and raised above the capsule; on section the nodules contained either a caseous or a purulent material, usually with a caseous, necrotic vermiform mass, evidently a degenerated immature worm; portal vein with thrombi, two of the latter with one immature kidney worm each; intima of veins roughened. Kidneys with several circular whitish spots, pyramidal in shape on section; no worms found in kidneys, perirenal fat or lymph-glands. Right anterior lobe of lung adherent to pleura, forming a pocket containing a mass of caseous material; a hemorrhage, about 2 cm. in diameter, adjacent to adhesion; lungs with several small, yellowish nodules filled with caseous material; small anterior lobe of one lung pneumonic.

Pig 18 was killed on May 12, 71 days after the last feeding of larvae. The postmortem results are given below:

Liver light brown in color, rather dull in appearance, but showing no evidence of congestion; surface of liver covered with fibrous threads and a small amount of fibrin; slightly elevated and rounded prominences present near edges of lobes; on section the prominences were found to be fibrous nodules, the centers of which contained a small quantity of purulent material; usually one immature kidney worm was present in each nodule; some of these nodules contained a sanguineous, purulent mass; a small percentage of the nodules contained caseous material but no worms; deeper portions of liver contained nodules similar to those present on surface; the latter nodules were situated along paths of the smaller branches of the portal vein; some fibrosis was noted in a few areas along margin of liver and in parenchyma; immature kidney worms present in portal vein and its branches, each worm being associated with a parietal thrombus. Several white spots visible on surface of kidneys; on section, these spots appeared to be old infarcts; kidneys and perirenal fat free from worms. With the exception of two atelectatic areas, each about 1 cm. in diameter, lungs appeared normal.

The data obtained from this experiment point to an invasion of the liver by way of the portal circulation, since both pigs showed immature worms in the portal vein, in association with thrombi. It is important to note that the deeper liver nodules in pig 18 were located along the paths of the smaller branches of the portal vein, which is interpreted as indicating that the larvae bored through these blood-vessels and that many of them became arrested and encapsulated, and some of them disintegrated in the connective tissue cysts for the proliferation of which they were responsible. The lungs of pig 17 were apparently invaded by larvae, as shown by the postmortem lesions, which were highly suggestive of those produced by these parasites. While the white spots noted on the surface of the kidneys of the pigs involved in these experiments might have resulted from the invasion of larvae, which could have reached these organs through

the systemic circulation, the failure to find live or degenerated worms in these lesions renders their interpretation rather doubtful.

Experiment II: On June 18, 1927, each of two pigs (19 and 20) from the same litter, 27 days old, was given a rich culture of infective kidney worm larvae by mouth. These animals were killed on August 30, 73 days after experimental feeding. The postmortem findings were as follows:

Pig 19: Liver pale in color and with an obvious thickening of perilobular connective tissue; surface of liver dull and roughened, due apparently to organization of fibrin deposits; many whitish nodules, about 0.5 cm. in diameter, protruding above level of liver capsule; larger nodules were felt on palpation of liver; nodules also found on section of liver, in most cases along course of portal vein and its branches; each nodule contained either a live worm and a small quantity of caseous material or a caseous brownish mass with a partly necrosed worm; there was some difficulty in cutting the liver, and on section the periportal tissue as well as the perilobular connective tissue was found to be noticeably increased; two immature kidney worms found under capsule on ventral surface of liver. Other organs and tissues apparently normal.

Pig 20: Liver similar in appearance to that of pig 19 and containing immature worms; increase in connective tissue more marked than in pig 19; portal vein showed three thrombi, each containing a live worm; one thrombus about 0.5 cm. long was located in portal vein. about 1 cm. from its bifurcation; another thrombus was located at the bifurcation, and the third thrombus was in one of the branches of the portal vein. Other organs and tissues apparently normal.

The results of this experiment are essentially similar to those of experiment I and point to an invasion of the liver by the worms through the portal circulation with resultant production of lesions including proliferation of connective tissue. It is important to note that two kidney worms were found under the liver capsule of pig 19. The significance of this will become apparent in connection with the data on the later experiments which are given in subsequent pages.

Experiment III: On February 10, 1928, each of 6 pigs from the same litter (21 to 26, inclusive), fairly uniform in size, and about 10 weeks old, received a culture of infective kidney worm larvae by mouth. Pigs 21 and 22 were killed on May 18, 1928, 88 days after experimental feeding. The postmortem records are given below:

Carcasses in good condition. Peritoneal covering of stomach and diaphragm very rough, due to the presence of an organized fibrinous exudate. Liver roughened and fibrous; perivascular connective tissue thickened and fibrous and containing encysted immature kidney worms; portal vein with numerous thrombi varying from the size of a pea to that of a hazelnut; intima of portal vein roughened and scarred; in certain areas perforations in the wall of the portal vein leading to cysts in the adventitious coat were noted; each of these cysts contained one immature kidney worm; gastro-hepatic artery

hard and irregularly dilated; on section it was noted that the lumen of this artery was almost entirely occluded by thrombi in various stages of organization; immature kidney worms found in association with thrombi in portal vein and gastro-hepatic artery. Two small nodules, apparently due to kidney worms, found in lungs of pig 21; lungs of pig 22, normal.

On June 8, 119 days after experimental feeding, pigs 23 and 24 were killed and examined postmortem. The lesions and worm infestations noted were very similar to those of pigs 21 and 22. The following lesions and worm infestations seen in these two pigs are of special significance, however:

Pig 23 showed several lung lesions. An incision into the lesions revealed a degenerated mass suggestive of kidney worms which had died and disintegrated; the lungs of pig 24 were normal. The pancreas of the latter pig showed a lesion suggestive of that produced by kidney worms; immature kidney worms were found in the portal vein and gastro-hepatic artery of both pigs. The renal areas of these pigs were free from infestation.

The remaining pigs of this lot (25 and 26) were killed on September 11, 214 days after experimental feeding of larvae. The results of the postmortem examinations for kidney worms and associated lesions were as follows:

Pig 25: Liver with some cirrhotic areas; on section, an occasional degenerated area was noted. The following lesions were noted in connection with the examination of the portal vein: perivascular connective tissue increased and containing immature kidney worms and caseous areas; intima of vein scarred and containing thrombi which were in the process of becoming organized and absorbed; in some areas a network of fibrous bands extended across the lumen of the vein; no kidney worms found in lumen of vein; walls of gastro-hepatic artery much thickened and containing small caseous areas; intima of artery badly scarred and showing thickenings which represent, apparently, organized thrombi. Kidney worms present in perirenal fat, in pelvis of kidney and in lumbar muscles.

Pig 26: Lesions in liver and portal vein similar to those of pig 25; perivascular tissue thickened; the thrombi noted in the portal vein were larger, however, than those noted in pig 25; two immature worms found in portal vein; two canals found in wall of portal vein communicating with cysts which contained immature worms; gastro-hepatic artery contained a thrombus which was becoming soft and showed evidence of organization; two immature worms found in this mass and one worm found perforating wall of gastro-hepatic artery. Kidney worms found in pelvis of kidney, in perirenal fat and in lumbar muscles.

The postmortem data of pigs 21 and 22 are in agreement with those of the previous experiments. It is important to note, however, that worms were found in these pigs in the gastro-hepatic artery, in association with thrombi, which indicates that some of the larvae which reached the lungs, either through the lymphatics or through the liver, probably by way of the liver, were returned to the heart and distributed by the systemic circulation. It is important also to note that the worms were found

perforating the portal vein of these pigs, and that the gastro-hepatic artery of pig 26 was found perforated by a worm. Pigs 23 and 24, killed 119 days after experimental feeding, were free from renal and perirenal infestation, as were pigs 21 and 22, killed one month earlier. Pigs 25 and 26, killed 214 days after experimental infestation, contained worms in the perirenal fat, the pelvis of the kidney, and the psoas muscles. These pigs still contained worms or lesions, or both, in the portal vein and gastro-hepatic artery, despite the fact that seven months had elapsed since these pigs had been infected. The data obtained in this experiment show conclusively that the renal region became infested relatively late in the course of the life cycle of the parasites, presumably as a result of a migration of the worms from some other part of the body to the renal organs and adjacent tissues.

Experiment IV: On October 19, 1928, each of two 12-day-old pigs (27 and 28) from the same litter was given a rich culture of infective kidney worm larvae by mouth. Pig 27 was killed three days after experimental feeding and no evidence of kidney-worm infestation could be demonstrated in the heart-blood, in press preparations of the portal vein, intestinal lymph-glands, lungs, liver and other organs and tissues commonly invaded by kidney worms.

Pig 28 died on January 5, 1929, 77 days after experimental feeding. The following lesions and worm infestations were noted:

About 300 cc of an orange-colored fluid was siphoned off from the peritoneal cavity; an approximately equal quantity of sanguineous fluid was siphoned off from the thoracic cavity; peritoneum roughened and covered with a fibrinous exudate; worms free on peritoneum and in peritoneal cavity close to kidneys; liver adherent to diaphragm; surfaces of liver covered with fibrinous exudate and pus, and containing immature worms; immature worms present under liver capsule, some of the worms in the process of wriggling through perforations in the capsule; in all latter cases observed, the anterior end of the worm was extruded through the perforation in the capsule; section through liver showed numerous lesions and worms throughout periportal tissue; worms found on peritoneal surface of diaphragm and in region of diaphragm adherent to liver; entire surface of spleen and mesenteries covered with fibrinous exudate; spleen adherent to diaphragm and immature worms present in area of adhesion of these two organs; worms abundant in area between liver and stomach. Wall of thoracic cavity roughened and covered with fibrinous exudate; an abscess containing immature worms, from one-quarter to one-half the size of those in the liver, found in lungs; one worm was found in trachea.

The failure to find evidence of infestation in pig 27 is by no means conclusive proof that the experimental feeding of this pig was a failure. The absence of macroscopic lesions is not

surprising, since only three days had elapsed from the time that the animal was infected to the date of necropsy. The failure to find larvae in press preparations of the liver and lungs might possibly be explained on the basis that the particular portions of these organs which were examined were free from infestation.

The postmortem data obtained in connection with pig 28 is significant in that it affords conclusive evidence of the perforation of the liver capsule by kidney worms. The worms which escaped from the liver came to lie on the surfaces of the abdominal organs, and elsewhere, such as the liver capsule, the diaphragm, in the area between the liver and the stomach and elsewhere in the peritoneal cavity. Once free in the abdominal cavity, the worms can readily reach the perirenal fat and penetrate this rather non-resistant tissue. In this connection it is important to note that worms were found free in the peritoneal cavity close to the renal region, and that no worms had as yet penetrated the perirenal fat. Pig 28 evidently suffered from a severe infestation to which it succumbed, as far as can be judged from the lesions noted at necropsy. The lesions found in the thoracic cavity of pig 28 are significant, since they show that massive infestations with kidney worms have greater potentialities of producing injury than has been previously suspected.

Experiment V: On October 24, 1928, a 17-day-old pig (29) was given a massive dose of infective kidney worm larvae by mouth. On November 13, 20 days after experimental feeding, the pig appeared sick and showed the following symptoms: Arched back, respiratory difficulty, particularly noisy breathing, a fast pulse, a subnormal temperature of 97.4° F., diarrhea, and weakness in the hind legs. On the following day the animal died. Postmortem examination revealed a bronchopneumonia. Grayish spots were found in liver and kidneys; larval kidney worms were observed in press preparations of the liver; larvae were obtained from the liver and lungs by digesting these organs in an acidified solution of scale pepsin, U. S. P. No larvae were recovered from the renal organs.

The severe symptoms shown by this pig were undoubtedly due to the fact that the animal was infected when only 17 days old, a period in life when it presumably had but little resistance to cope with parasites which become so widely distributed to various parts of the body. It indicates, moreover, that massive infestation with kidney-worm larvae acquired under normal conditions may be a cause of death in young pigs, and that this cause might easily be overlooked, since lesions more character-

istic of kidney-worm infestation than those found in this animal apparently do not develop in the course of 17 days.

Experiment VII: On October 27, 1928, each of three 20-day-old pigs (30, 31 and 32) from the same litter was given a rich culture of infected kidney-worm larvae by mouth. Pig 30 was killed on November 23, 27 days after infection. Postmortem examination revealed the following:

Animal markedly emaciated; body fat gelatinous. Liver very pale, with necrotic tracks under the capsule and some fibrinous exudate on the capsule; a small parietal thrombus, containing an immature worm, was noted in one branch of portal vein. Kidneys with pale circumscribed areas; kidney-worm larvae found in press preparations of liver but not in those of kidneys. Lungs with many hemorrhagic spots and small pneumonic areas more or less evenly distributed; kidney-worm larvae found in press preparations of lungs.

Pig 31 died on December 4, 38 days after experimental infection. The following lesions and worm infestations were noted:

Carcass emaciated; slight increase in amount of abdominal fluid. Liver showed characteristic lesions and, in addition, contained three markedly elevated areas about the size of a small pea, each of these areas containing an immature worm, located immediately under the capsule; lumen of portal vein almost occluded by a thrombus which extended into the large branches of that blood-vessel. Kidneys apparently normal. Lungs slightly edematous and congested; a few small, pinhead-sized, pneumonic areas evenly distributed over posterior lobes; kidney-worm larvae found in press preparations of pulmonary tissue taken from these areas.

Pig 32 died on December 10, 44 days after experimental feeding. Postmortem examination revealed the following lesions and worm infestations:

Carcass emaciated. Liver similar to that of pig 15; lumen of portal vein almost entirely occluded by yellowish thrombus extending over the entire length of main trunk; periportal connective tissue noticeably increased. Kidneys with numerous pale spots, but no larvae demonstrable in press preparations. Lungs slightly edematous and congested; few small pneumonic areas scattered throughout lung tissue, especially in posterior lobes; kidney-worm larvae found in press preparations of pneumonic areas; vena cava normal.

The pneumonic areas noted in the three pigs involved in these experiments were associated with kidney-worm larvae in all cases. Whether this condition was due to the fact that the pigs used in these experiments were only about three weeks old and the capillaries of the lungs were too small to permit the larvae to pass through, or whether the larvae became arrested in the lungs for some other reason, cannot be determined on the basis of available information. It is clear, however, that when pigs acquire an infestation with kidney worms at an early age, they are likely to become seriously affected and to succumb, as did two out of the three pigs involved in this experiment. In pigs of that age, succumbing to a natural infestation with kidney

worms, the cause of death might easily be overlooked unless precautions were taken to make a thorough examination for larvae. It is interesting to note that the kidneys of pigs 30 and 32 showed lesions, in the form of superficial circumscribed areas, and that no worm larvae were demonstrable in these lesions. Assuming that the lesions in question were produced by kidney-worm larvae which reached the kidneys through the systemic circulation, the larvae at this early stage are apparently ill-adapted to survive in the renal tissue and apparently perish in that location before they undergo any conspicuous growth and development. The lesions in the liver and in the portal vein noted in these pigs were typical of those usually noted in cases of experimental stephanuriasis.

The following experiments were carried out with definite numbers of larvae administered to pigs by mouth, in order to determine whether there is a relation between the number of larvae administered and the severity of the lesions which develop.

Experiment VIII: A 39-day-old pig (35) was fed 5,000 infective larvae on March 16. This animal died on April 9, 24 days after experimental infection. On postmortem examination it was found that death was due to an acute fibrinous peritonitis; the ileum was dark in color in a section about two inches long, necrosed and ruptured, with its contents escaping into the peritoneal cavity. While kidney-worm larvae were not demonstrated in the liver, sections through this organ revealed characteristic lesions produced by these parasites.

A 40-day-old pig (34), a litter mate of pig 33, was fed 2,500 larvae on March 17, 1929. This animal died on June 12, 87 days after experimental feeding. The postmortem findings were as follows:

> Carcass markedly emaciated. Abdominal cavity contained a greatly increased quantity of fluid which was sero-purulent. Liver roughened, adherent to diaphragm, covered with a fibrinous exudate, and containing immature worms on its surface; many worms visible under liver capsule and several found perforating the capsule; on section liver showed increase in interlobular connective tissue, necrotic areas and numerous immature worms; portal vein with numerous thrombi and small worms; periportal connective tissue and tissue around portal lymph-gland thickened and containing numerous worms; spleen covered with fibrinous exudate. One immature worm found adhering to omentum. Kidneys with white spots and scars but no worms in renal organs and in perirenal fat. Lungs covered with a fibrinous exudate, and containing nodules filled with pus; posterior vena cava thrombotic and containing scars in areas which worms had apparently perforated.

A 39-day-old pig (35) from the same litter as pigs 33 and 34 received 1,500 *Stephanurus* larvae on March 16. This animal was killed July 31, 137 days after experimental infection. The

liver showed characteristic worm lesions and worms in the process of perforating and emerging through the capsule; the portal vein was thrombotic and five immature worms, associated with thrombi, were recovered from this blood-vessel. One worm was found embedded in the diaphragm and 12 incompletely grown worms were recovered from the perirenal fat; there was no evidence of peritonitis.

The death of pig 33, 24 days after experimental infection, was due to a severe peritonitis following necrosis and rupture of the ileum, a condition apparently due to something other than kidney-worm infestation. Pig 34, which died 87 days after experimental infection, showed lesions essentially similar to those noted in pig 28. Worms were found in liver tissue immediately underneath the capsule and some worms were perforating the capsule; the worms free on the surface of the liver had apparently succeeded in breaking through the capsule. The occurrence of worm lesions in the posterior vena cava is significant in that it shows the path of migration of the worms from the liver to the lungs.

Further proof of the ability of immature kidney worms to perforate the wall of a blood-vessel is afforded by the finding of scars in the wall of the vena cava of this pig. Pig 35, killed 137 days after experimental infection, showed 12 incompletely grown worms in the kidney fat; presumably these worms had reached the perirenal fat after having perforated the liver capsule. Apparently the worm which was found embedded in the diaphragm also came from the liver. It is interesting to note that pig 35, which received the smallest number of larvae, survived, whereas the two other pigs died, the death of pig 34 being due, apparently, to the worm infestation.

Another experiment, involving the feeding of definite numbers of larvae, was carried out as follows:

Experiment IX: On July 6, 1929, three pigs (36, 37 and 38) were given kidney-worm larvae by mouth as follows: Pig 36 received 3,400 larvae, pig 37 received 2,500 larvae, and pig 38 received 1,700 larvae. These pigs were killed on April 8, 1930, 9 months 2 days following experimental feeding. The postmortem findings were as follows:

Pig 36: Liver adherent to diaphragm and showing numerous scars, healed for the most part, though some lesions still showed pus and degenerated worms on section; portal vein scarred and lumen occluded with thrombi; periportal tissue thickened and containing worms; other hepatic blood-vessels with thrombi. Pelvis of left kidney distended and containing considerable pus; pelvis of right kidney normal; ureters with pus and showing several openings in the wall, apparently due to kidney worms; one kidney worm found in ureter and numerous kidney

worms present in cysts in perirenal fat along course of ureter; numerous worms present in lumbar muscles, close to kidneys. Lungs with several greenish nodules containing pus and incompletely grown kidney worms; kidney worms noted in thoracic cavity in region of vertebrae.

Pig 37: Liver showed a moderate number of healed scars; branches of portal vein thrombosed; periportal connective tissue thickened and containing a few worms; several organized lesions on diaphragm; lesions containing worms in area between liver and diaphragm. Right kidney normal on superficial appearance, and containing two worms in pelvis and a small parenchymatous retention cyst; left kidney with pronounced scars on cortex and containing a small retention cyst; two worms found in wall of one ureter; perirenal fat containing kidney-worm cysts free from worms; an opening from a worm cyst into the ureter was definitely traced. Lungs with old, healed lesions.

Pig 38: Liver showed a few healed and no active lesions; periportal connective tissue thickened and abscessed; abscesses contained half-grown kidney worms. Left kidney with scar in capsule; kidney fat surrounding left kidney contained numerous worms; one worm in pelvis of right kidney; numerous worm cysts along course of ureter, some of them opening into lumen of ureter; one worm found perforating ureter from cyst, the anterior half of the worm being in the ureter and the posterior half being in the periureteral cyst. Two lesions noted in lungs, one of them containing a half-grown kidney worm.

It is important to note that the three pigs involved in this experiment, which were killed nine months after experimental feeding, showed worms in the perirenal fat, in the kidneys and in the ureters. It is important to note also that pigs 37 and 38 still showed worms in the act of perforating the wall of the ureter, indicating that some of the parasites had succeeded in extricating themselves from the liver late in the course of their development. It shows also that worms continued to migrate to the renal region over a long period. This probably accounts in part for the fact that up to a certain age pigs from kidney-worm-infested stock tend to show heavier renal infestation as they grow older. Most of the liver lesions noted in these pigs were healed and no longer contained live worms. The lung lesions had not as yet healed and incompletely developed worms were still present in the lungs of two pigs. The worms which persisted in the periportal connective tissue were incompletely grown. In a general way the degree of reaction to the infestation was proportional to the number of larvae ingested.

On the basis of the available evidence, it is safe to conclude that, having invaded the perirenal fat, the as yet incompletely devloped kidney worms burrow into the fatty tissue and establish channels to the ureters which they perforate. From this location the worms can reach the pelvis of the kidney.

Owing to their rooting habits, the possibility that pigs might acquire an infection with *Stephanurus* through the nasal route

cannot be entirely overlooked. To test this possibility, the following experiment was carried out:

Experiment X: On June 29, 1928, a rich culture of infective kidney-worm larvae was placed, by means of a pipette, in the nasal cavity of each of four pigs (39 to 42, inclusive). Pig 39 was killed on September 12, 75 days after attempted experimental infection feeding through the nose. The postmortem results were as follows:

Carcass in good condition. Surface of liver with scarred areas typical of those produced by kidney worms; portal vein thrombotic; immature worms present in perivascular tissue; gastro-hepatic artery roughened, thrombotic and filled with purulent material; one immature worm, about one-fourth grown, recovered from gastro-hepatic artery. Kidneys and perirenal fat appeared normal and free from worms. Lungs normal.

Pig 40 was killed on October 9, 102 days after experimental infection. This animal showed but very few liver lesions and no lesions were noted in the portal vein or in any other blood-vessels. Other organs and tissues normal.

Pig 41 was killed October 22, 125 days after experimental infection. The following lesions and worm infestations were noted at necropsy:

Organized fibrinous exudate on stomach wall, diaphragm and part of spleen; immature kidney worms found free in peritoneal cavity, in connective tissue between stomach and liver, and one immature worm found free on the surface of right kidney; liver with scarred areas; portal vein and gastro-hepatic artery thrombotic; two immature worms found in one partially organized thrombus of gastro-hepatic artery which practically occluded the lumen of this blood-vessel. Except for the presence of a worm on surface of right kidney, renal area including the perirenal fat free from lesions and worms. Lungs without lesions.

Pig 42 was killed December 20, 174 days after experimental infection. The following lesions and worm infestations were noted:

Liver with typical scarred areas of varying sizes; one liver abscess about the size of a walnut filled with a creamy pus and containing one live, half-grown worm. A few worms found in perirenal fat; one worm found in pelvis of kidney. Lungs normal.

The results of this experiment are in harmony with experiments involving the feeding of larvae by mouth. Presumably the worms, on getting into the nares, became activated by the heat of the pig's body and probably wriggled down until they reached the pharynx and were swallowed; the possibility that the larvae might have penetrated the nasal mucosa and thus reached the blood-stream cannot, however, be excluded. In connection with the postmortem data, it is interesting to note that the lesions noted in pig 39, killed 75 days after experimental

infection, were essentially similar to those noted in other pigs on necropsy at a corresponding stage in the course of experimental infection. Pig 40 evidently escaped infection for the most part; pig 41, killed 125 days after experimental infection, showed characteristic lesions and worms still free in the abdominal cavity; the renal region of this pig was free from worms and lesions. Pig 42, killed 174 days after experimental feeding, showed worms in the renal region, as did other pigs killed at a more or less corresponding stage of infestation following feeding of larvae by mouth.

Experimental Infection of Guinea Pigs

In order to obtain data on the path of migration of the early-stage larvae to the various organs and tissues and to discover, if possible, the location of the larvae within a few days after they have been swallowed, the following experiments were carried out, using guinea pigs as host animals.

Experiment XI: The following experiment was carried out by one of us (Schwartz):

A guinea pig was fed an undetermined number of infective *Stephanurus* larvae on March 12, 1927; the animal was examined postmortem on April 14, 33 days after experimental infection, and a number of larvae were recovered from the mesenteric lymph-glands; examination of the liver and lungs in press preparation failed to reveal larvae.

Additional experiments with guinea pigs were carried out by Joseph E. Alicata, Junior Zoölogist in the Bureau of Animal Industry, under the direction of the senior author, as follows:

Experiment XII: Each of three guinea pigs (1, 2 and 3) was given several hundred infective larvae by mouth, on December 1, 1928.

Guinea pig 1 was killed on December 6 with the following postmortem findings: The peritoneal fluid was siphoned off and found to be free from larvae; one larva was found in a press preparation of the spleen; no larvae were found in press preparations of the pancreas; eight larvae were found in a number of press preparations of the mesenteric lymph-glands, particularly those in the region of the cecum; one larva obtained from a lymph-gland was 515μ long by 27μ in maximum width, indicating little or no growth beyond that attained by the infective larva. One larva was contained in the clotted blood obtained from the cavity of the heart; the lungs showed a number of petechial hemorrhages and four larvae were found in a number of press preparations of this organ.

Guinea pig 2 was killed on December 10. The peritoneal fluid was free from larvae; the liver showed several small grayish spots but no larvae were seen in press preparations of this organ; press preparations of the spleen and pancreas revealed no larvae; six larvae were found in several press preparations of the cecal lymph-glands; press

preparations of the kidneys showed no larvae. The heart-blood was free from larvae.

Guinea pig 3 was killed December 18. The peritoneal fluid, liver, spleen, pancreas, and kidney were negative for larvae; the liver showed spots suggestive of kidney-worm infestation; four larvae were found in several press preparations of the mesenteric lymph-glands. The heart-blood was free from larvae; the lungs showed spots suggestive of kidney-worm infestation but no larvae were seen in press preparations of these organs.

Experiment XIII: Each of three guinea pigs (4, 5 and 6) was given 1,000 infective *Stephanurus* larvae by mouth, on March 26, 1929.

Guinea pig 4 died on May 6, 41 days after experimental infection. Postmortem examination revealed liver lesions suggestive of those produced by kidney worms, but no larvae were found in press preparations of this organ; mesenteric lymph-glands and spleen were examined in press preparations without larvae being found; no larvae were found in the lungs or heart-blood. The kidneys contained no larvae.

Guinea pig 5 died May 21, 56 days after experimental infestation. Several whitish spots were seen on surface of liver; one immature kidney worm, 17 mm. long by 1 mm. wide, was recovered from the liver; the pancreas, spleen, and omentum showed lesions and blood clots; 3 larvae were recovered from the pancreas and spleen in press preparations. The latter measured as follows: 1.8 mm. long by 77μ wide; 4 mm. long by 325μ wide; 1.3 mm. long by 93μ wide. Other organs normal.

Guinea pig 6 was killed October 23; no larvae and no lesions were noted at necropsy.

The data obtained in connection with experiments on guinea pigs are in the main confirmatory of those obtained in connection with the feeding of larvae to pigs. The occurrence of larvae in the mesenteric lymph-glands of experimentally infected guinea pigs warrants the inference that, having reached the intestine, the larvae penetrated its wall until they reached the lymph-spaces, from which they were carried in the lymphatics to the lymph-nodes, where some of them became arrested, at least for a time. The larvae in the lymph-nodes might penetrate the venous capillaries and thus be carried to the liver by the portal circulation, or they might continue in the lymphatics and thus reach the thoracic duct and be carried to the jugular vein and thence through the anterior vena cava to the right side of the heart, from which they would naturally get to the lungs by way of the pulmonary arteries. Other larvae penetrating the wall of the intestine might bore into the capillaries and thus enter the portal system directly and be carried to the liver, from which some of them might possibly continue with the circulation and reach the lungs by way of the hepatic vein, posterior vena cava and the heart. Some larvae were arrested in the lungs, as shown not only in experiments with guinea pigs 1 and 2, but also in

experiments with pigs. In the latter animals the larvae persisted in the lungs for many months, though their growth was not completed there. Some of the larvae which reached the lungs of guinea pigs apparently got through the capillaries of these organs and were returned to the left side of the heart and distributed by the arterial circulation to various parts of the body, including the spleen and kidneys, as already noted by the writers in a previous paper.[5] It is interesting to note that larvae were found in the spleen of guinea pig 1, and lesions suggestive of those produced by kidney-worm larvae were found in the pancreas, spleen and omentum of guinea pig 5. No larvae were found in the kidneys of guinea pigs.

Experimental Infection of a Calf

The occurrence of immature forms of *Stephanurus dentatus* in cattle (*Bos taurus*) was first noted by Hall.[3] Schwartz and Alicata[4] reported additional cases of *Stephanurus* infection in cattle in the United States, all specimens being incompletely grown and sexually immature. Since the publication of the latter report, considerable data have accumulated in the Zoölogical Division which show that southern cattle are frequently infested with this swine parasite. In view of this, it appeared desirable to determine experimentally the development of *S. dentatus* in a bovine. One experiment was carried out as follows:

Experiment XIV: On January 30, 1929, about 1,000 infective *Stephanurus* larvae were given to a calf by mouth. This animal was killed on September 5, 7 months 6 days following experimental feeding. The postmortem results were as follows:

> Surface of liver with a moderate number of scars, about 0.5 cm. in diameter, similar to those found in experimentally infected pigs; pancreas adherent to liver; one-half grown kidney worm found at site of adhesion; three thrombi in portal vein; an immature kidney worm found in one thrombus; a few worms found encysted in periportal tissue; several thrombi in gastro-hepatic artery; no worms in renal region. Wall of posterior vena cava scarred; scars led to a fistulous tract going through connective tissue and ending in adhesion between pancreas and liver where worm was found.

The results of this experiment are in harmony with what is known regarding the distribution of *S. dentatus* in the various organs and tissues of cattle, in which animals the worms usually fail to reach the renal region and consequently do not develop to fertile maturity. In this connection it should be noted, however, that Spindler[7] reported an immature kidney worm in the kidney region of a year-old calf slaughtered in Moultrie, Ga.

The worm was located near one of the kidneys, about 15 mm. from the renal lymph-glands.

DISCUSSION

The life cycle: The data presented in the foregoing pages show that the course of infection of pigs with *Stephanurus dentatus*, acquired as a result of swallowing the infective larvae, is similar to that which follows from experimental percutaneous infection, as shown by the data presented by the writers in a previous paper. Following ingestion of the larvae, the worms and associated lesions appeared in the liver and in blood-vessels entering and leaving this organ long before the parasites were demonstrable in the perirenal fat, ureters, kidneys, and psoas muscle. Hepatic infestation with accompanying lesions preceded renal infestation uniformly and consistently, which fact alone warrants the conclusion that the worms, which appear in the renal region, as they commonly do, relatively late in the course of the life cycle of the parasites, have reached that location as a result of a migration from some other organ or tissue. Presumably the worms have to undergo their earlier development outside of the perirenal fat and renal organs.

Direct and conclusive evidence of a transfer of the as yet incompletely developed kidney worms from the liver, and possibly from other abdominal organs to the renal region, is afforded by the data presented in this paper as well as by the data presented in connection with experimental percutaneous infections, which show that the worms which get into the liver by way of the circulation leave the portal vein and its branches and invade the hepatic tissue in which they migrate, ultimately coming in contact with the capsule. Their migrations immediately underneath the capsule are shown by well-marked linear tracks. Sooner or later, some of the worms perforate the liver capsule, thus entering the peritoneal cavity, in which they wander freely for a time on the surfaces of the organs and tissues. During their active migrations in the peritoneal cavity, the worms come in contact with the renal organs (see protocol on pig 38). The worms which come in contact with the perirenal fat penetrate this rather non-resistant tissue and ultimately become encapsulated there.

The ability of *Stephanurus* to penetrate tissues is one of the most striking features of its behavior in the bodies of susceptible hosts. Data presented in this paper show that in addition to penetrating various soft tissues, these parasites perforate the walls of blood-vessels, including the portal vein, which accounts,

in part, at least, for their accumulation in the periportal connective tissue; data also have been presented which point to the perforation by the worms of the wall of the vena cava of a calf as well as that of pigs. Having penetrated the perirenal fat, the parasites apparently continue their migrations within the perirenal fat and reach the wall of the ureters, which they perforate, as shown by the postmortem data presented in this paper. This affords an outlet for the eggs which are discharged to the outside with the urine. The worms which remain in the blood-vessels, in the hepatic tissue, in the psoas muscles, which they apparently perforate readily, and in other abdominal organs exclusive of the renal organs, do not complete the life cycle, since they either fail to develop to fertile maturity or have no means of expelling the eggs to the outside of the host's body. A similar fate awaits the larvae which fail to extricate themselves from the pulmonary capillaries and thus become arrested and subsequently encapsulated in the lungs, from which they may occasionally escape and come to lie free in the thoracic cavity.

Bauche and Bernard[1] were of the opinion that infection of pigs through the oral route, with consequent hepatic involvement, did not lead to the completion of the life cycle of *Stephanurus*. However, as already noted in the introductory statement of this paper, these investigators killed the experiment pigs, to which they had administered larvae by mouth, not later than 100 days following experimental feeding, and failed to find evidence of renal involvement. The data presented in this paper show that the worms tend to invade the renal region later than 100 days after experimental feeding. Thus, pigs 23 and 24, examined postmortem 137 days after experimental feeding of larvae, showed a few unencysted and immature worms in the perirenal fat, while pig 41, examined postmortem 125 days after experimental infection through the nasal cavities, was free from worms in the renal area. Pig 42, killed 174 days after experimental infection through the nasal cavities, showed renal involvement, as did pigs 25 and 26, killed 214 days after experimental feeding, and other pigs killed at relatively later stages in the course of infection. The earliest periods in which worms were found in the renal region of pigs following experimental percutaneous infections were 107, 112 and 113 days, respectively. On the other hand, two pigs killed 99 and 102 days after experimental percutaneous infection failed to show evidence of renal or perirenal involvement. The data obtained in experiment IV show that while the worms occurred free in the peri-

toneal cavity as early as 77 days after ingestion of larvae, the parasites did not penetrate the perirenal fat and renal organs in this relatively early stage of their development.

Lesions Associated with Kidney-Worm Infestation

The pathology associated with experimental kidney-worm infestation was essentially the same regardless of the portal of entry for the larvae, as shown by the protocols presented by the writers in this and in a previous paper.[5] The lesions observed in these cases were in all respects the same as those observed in cases of infestation acquired under field conditions, which have been described in considerable detail by Boynton.[2]

Lesions associated with kidney worms differ from those produced by many other nematodes which invade the tissues during their migrations in the course of their life cycle in that the former are of an acute inflammatory nature suggestive of being the result of pathogenic bacteria rather than resulting from the presence of the worms.

Peritonitis of varying degrees was observed in practically all pigs used in these experiments. In the majority of cases peritonitis was evidenced by a thickening and roughening of the peritoneal covering of the liver and adjacent structures, and by adhesion of the liver to the diaphragm. This condition was apparently due to an organization of a fibrinous exudate, indicating that an acute fibrinous peritonitis had occurred some time earlier, presumably when the larvae were actively migrating from the liver. In three cases (pigs 18, 28 and 34) deposits of fibrin were found on the liver and adjacent structures. In pig 18 the fibrin was partially organized and no worms were found on the liver surface or penetrating the liver capsule; in pigs 28 and 34 the condition was more acute, and immature worms were found penetrating the liver capsule and in the fibrin deposits.

The occurrence of an exudative peritonitis in kidney-worm infestation is not readily explained. However, three possibilities are suggested, namely: (1) puncture of the stomach or intestine by migrating worms; (2) contamination of the serous surfaces and tissues with bacteria brought in with the larvae during their migration from the intestine in cases of infection by the oral route; and (3) contamination of the peritoneum with pathogenic bacteria which are frequently present in apparently normal livers, and which might reach the peritoneum through punc-

tures of the liver capsule during the migration of the worms from the liver.

The first of these possible explanations appears to be the most logical since there is some evidence to support it. In a previous paper[5] the writers reported the occurrence of worms in five instances under the peritoneal covering or partially penetrating the wall of the stomach, and in one instance a worm was found partially penetrating the wall of the duodenum. While no worms that had succeeded in perforating the wall of these organs were found, it is possible that they might have been overlooked or had become dislodged during necropsy.

The formation of abscesses in the organs and tissues invaded by kidney worms is apparently due to bacteria carried to these locations by the migrating worms. While no bacteriological study was made by the writers to determine this point, this supposition is supported by observations of Shealy and Sanders,[6] who reported the finding of "various pus-producing organisms" on microscopic examination of pus from kidney-worm lesions.

Thrombus formation in the portal vein, gastro-hepatic artery, and posterior vena cava is due apparently to the injuries to the intima produced by worms penetrating the vessels from within, or by worms in the adventitious coat which possibly succeed in perforating the wall of the vessels from without. These injuries furnish favorable locations for the accumulation of leucocytes which, upon disintegration, liberate the necessary ferments to bring about the deposition of fibrin and ultimate formation of thrombi.

Despite the finding of thrombi in the gastro-hepatic artery in the majority of pigs used in the experiments recorded in this paper, as well as those reported in the writers' previous paper,[5] no case of involvement of the larger arteries or the formation of aneurysms, such as were reported by Boynton,[2] was observed.

Acute exudative pleurisy of the sero-fibrinous type, such as observed in pig 28, appeared to have resulted from an extension of the inflammatory process from the peritoneal cavity, since this was accompanied by a peritonitis characterized by a similar type of exudate.

Summary and Conclusions

The principal facts and discussions presented in this paper may be summarized briefly as follows:

Following experimental feeding of infective larvae of *Steph-*

anurus dentatus to pigs, the worms were distributed by the circulation to various organs and tissues and became localized principally in the portal vein and in its branches, the periportal tissue, the gastro-hepatic artery, the hepatic tissue, and the lungs. The perirenal fat, the ureters and kidneys, and the psoas muscles became infested relatively late in the course of the life cycle of the parasites.

Five pigs, killed from 20 to 48 days following experimental feeding of larvae, showed liver and lung involvement in the form of lesions and worm infestations; 8 pigs, killed from 71 to 88 days following experimental infection by mouth, showed liver involvement, and all but one liver contained worms, while only 4 animals of this series showed lung involvement, worms being actually present in the lungs of only one pig; of 10 pigs killed from 119 days to somewhat over 9 months following infection by the oral route, all showed liver involvement with worms still present in most cases, and only 4 showed lung involvement. Three pigs of the last group, killed over 9 months after experimental infection, showed lung involvement with incompletely developed live worms still present in two pigs.

Though worms were absent from the renal organs and perirenal fat of one pig killed 125 days after experimental feeding of larvae, incompletely developed worms were found in the perirenal fat of another pig killed 137 days after infection by mouth; in experimental percutaneous infection the presence of worms in the perirenal fat was noted in one pig as early as 107 days following subcutaneous injection of larvae, while another pig, killed 102 days after subcutaneous injection of larvae, contained no worms in the perirenal fat and was otherwise free from renal infestation.

The consistent occurrence of worms or worm lesions, or both, in the liver of pigs, following the administration of *Stephanurus* larvae by mouth, indicates that this organ, though actually a temporary habitat of these parasites, is in their path of migration and leads to their final destination, namely, the renal region; the latter is the only location that affords an outlet for the eggs to the outside world. The lungs are an accidental habitat of *Stephanurus dentatus* which, dependent upon the lymph and blood circulation for their transportation from the wall of the intestine to the liver, are frequently arrested in the capillaries of the lungs to which they are brought by way of the lymphatic system and also by way of the liver. The worms which become arrested in the lungs, however, fail to attain their full development, a fate which also overtakes those worms which become

encapsulated in the liver, in the periportal tissue and elsewhere, other than in the renal region. A similar fate awaits the worms which become arrested in blood-vessels where they are invariably associated with thrombi.

The available evidence points to an invasion of the liver by the larvae which have been swallowed, principally by way of the portal circulation, as evidenced by the almost constant occurrence of incompletely developed worms in the portal vein and in its branches. The data presented in this paper also indicate that practically all the larvae which reached the liver were arrested in the capillary network of that organ. The occurrence of larvae in the gastro-hepatic artery points to an invasion of the liver by larvae carried in the systemic circulation.

Though worms were not found in the perirenal fat earlier than 137 days after the ingestion of larvae, the liver capsule of pigs killed as early as 77 and 87 days after infection by mouth, showed evidence of having been perforated by worms; some of the latter were found perforating the capsule and others were found free on the surface of the liver and elsewhere in the abdominal cavity.

Though pigs killed in the relatively early stages of infection, and long before the worms were encountered in the perirenal fat, frequently showed renal lesions which might possibly have been the result of an invasion by *Stephanurus* larvae which reached the kidneys through the systemic circulation, the lesions in question were superficial, circumscribed areas, free from worms in all cases.

The available evidence indicates that the worms found in the kidneys and ureters of the experimentally infected pigs reached these organs by way of the perirenal fat which the parasites would have no difficulty in penetrating, judging by their capacity to perforate the more resistant liver capsule and the rather tough walls of blood-vessels. That the worms which penetrated the perirenal fat reached this tissue from the peritoneal cavity in which they wandered freely after their liberation from the liver is a conclusion which is fully justified by the data presented in this and in a previous paper on experimental percutaneous infection. Worms were not found free in the peritoneal cavity and on the surfaces of its organs before the liver capsule had been ruptured; neither were worms found in the perirenal fat at any time before the worms succeeded, or could have succeeded, in extricating themselves from the hepatic tissue, as shown by the protocols presented in this and in a previous paper on percutaneous infection.

The available evidence warrants the inference that worms which escaped from the liver as a result of perforating its capsule constituted the principal, if not the only, source of parasites which invaded the perirenal fat. In the latter tissue the worms evidently continued burrowing, thus establishing pathways which led to the ureters; the worms perforated the walls of these ducts, as shown conclusively by the data presented in this paper. The perforation of the wall of the ureters enabled the worms to enter these ducts, in the walls of which they might become encapsulated as they apparently did on several occasions, and in the lumen of which they could wander, thus reaching the pelvis of the kidney. It is evident that occasionally worms might migrate in the renal tissue and come to lie immediately under its capsule, as shown in connection with the protocols presented in this and in a previous paper.

Aside from perforating the liver capsule, the worms evidently punctured the walls of the blood-vessels in which they were commonly encountered, as shown by the finding of scars in the walls of these vessels, of canals leading to adventitious cysts in the portal vein, and of worms in the process of perforating the wall of this blood-vessel. This accounts, in all probability, for the accumulation and ultimate encystment of the worms in the periportal connective tissue and for the occurrence of worms, free and encysted, in the hepatic tissue, the cysts occurring principally along the course of the branches of the portal vein.

The data presented in this paper invalidate the conclusions of Bernard and Bauche,[1] who, on the basis of postmortem findings on pigs infected with *Stephanurus* larvae by mouth and killed at various intervals, but not later than 100 days following experimental infection, inferred that infection by the oral route resulted in hepatic infestation but not in renal infestation.

The data obtained in connection with experimental infection of guinea pigs have thrown light on the paths of migration of *Stephanurus* larvae from the wall of the intestine to the liver, lungs, and other organs in which they have been commonly encountered. The occurrence of the larvae in the mesenteric lymph-nodes affords conclusive proof of their invasion of the lymph-stream, from which they could naturally reach the lungs. The larvae, once arrested in the lymph-nodes, might also reach the liver by penetrating the venous capillaries in the nodes and thus get into the portal circulation. The protocols on guinea pigs have also afforded direct evidence of the passage of the larvae through the heart, as shown by the finding of a larva in the heart-blood of one of these animals, and have shown

moreover that the larvae can reach the spleen and pancreas, presumably through the systemic circulation. Though larvae were found in various locations in guinea pigs at various intervals following infection by mouth, they were absent from the renal tissue of these animals, indicating that these worms are not adapted to survive in the renal organs at the time that they might be brought there by the systemic circulation.

A calf was experimentally infected with *Stephanurus* as a result of administering larvae by mouth; though this animal was killed and examined postmortem more than seven months after experimental infection, a period adequate to bring about renal infestation in pigs, the calf was entirely free from renal lesions and infestation, but showed hepatic involvement. This finding is in harmony with the known facts regarding the distribution of *Stephanurus dentatus* in bovines, in which hosts the worms usually occur in the liver in association with typical lesions, and seldom succeed in invading the renal region.

Stephanurus dentatus, in common with species of *Strongylus* parasitic in horses, occupies a more or less unusual position among nematodes, as far as its life history is concerned, in that the worms commonly get into various locations, blind alleys for the most part, from which they cannot extricate themselves and in which they are destined to perish without completing their development. The worms which become arrested and encapsulated in the lungs, in the liver, in the periportal tissue, in the psoas muscles, on the diaphragm and elsewhere in the body, excepting the renal organs and perirenal fat, though capable of surviving for long periods, are to be regarded as erratic worms against which the body has built up its defenses in the form of firm cysts before the worms were able to continue along the paths which lead to the completion of their life cycle.

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THE INCIDENCE OF BANG ABORTION DISEASE IN TUBERCULOUS CATTLE*

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Gilman[1] has pointed out that lesions due to *Brucella abortus* have been found in guinea pigs which had been injected with milk suspected of containing tubercle bacilli. He suspects that in some of the earlier investigations these lesions must frequently have been confused with tuberculosis. This suggests that in the past, when tuberculosis was more common in cattle, both tuberculosis and Bang's disease may have been present in the same herd if not in the same individual.

N. Plum[2] and A. Thomsen[3] have found both *Br. abortus* and acid-fast organisms in smears from placenta of animals that had aborted. They state that tuberculosis and abortion infection may be found together. The acid-fast organisms were found in only 1.4 per cent of the cases examined, so one might gain the impression that both infections occur rarely in the same animal. Lesions of tuberculosis are rarely encountered in the uteri of reactors to the tuberculin test but are commonly found in the lungs and lymph-glands of such animals. Infection with Bang's disease is largely confined to the uterus and udder. It would therefore seem reasonable to assume that an examination of the placenta would fail to reveal many cases where both infections are present.

This investigation was undertaken to determine if infection with *Br. abortus* and tuberculosis in the same individual is a rare or a common occurrence.

SOURCES OF DATA

Arrangements were made by Dr. E. T. Faulder, of the Department of Agriculture and Markets, for obtaining blood samples from reactors to the tuberculin test. A box containing sterile bottles was sent each week to Dr. J. W. Claris, in charge of the Buffalo office of this Department. Following Dr. Claris' suggestion, the bottles were distributed among the veterinarians making the postmortem examinations in such a manner that a few samples were obtained from each group of reactors. It was thought that the use of this method would give a total more

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TABLE I—*Agglutination test and postmortem records.*

Sample	Reject Tag Number of Animal	Agglutination Test Dilutions						Postmortem Lesions					
		1:25	1:50	1:100	1:200	1:400	1:800	Retro-pharyngeal	Bronchial	Mediastinal	Lungs	Mesenteric	Other Organs
241	49694	+	+	+	+	–	–	–	XX	X	–	X	–
242	8842	+	–	–	–	–	–			NVL			
243	8841	+	+	+	+	+	+	–	XX	–	–	–	–
244	8847	+	–	–	–	–	–	–	X	–	–	–	–
245	8846	+	–	–	–	–	–	XX	–	–	–	–	–
246	8834	+	+	–	–	–	–	–	–	XX	–	X	–
247	8845	+	–	–	–	–	–	–	–	–	–	XX	–
248	8843	+	P	–	–	–	–	–	XX	–	–	–	–
249	49695	+	+	+	+	+	+	–	XX	X	–	–	–
250	56776	+	P	–	–	–	–			NVL			
251	16452	+	+	+	–	–	–	–	–	X	X	–	–
252	56713	+	+	–	–	–	–			NVL			
253	56704	+	+	+	+	+	+	X	–	–	X	–	–
254	56702	+	+	–	–	–	–	–	X	X	–	X	X
255	56794	+	+	–	–	–	–	–	X	X	–	–	XX
256	56705	+	–	–	–	–	–	X	–	X	–	–	–

TABLE I—*Agglutination test and postmortem records—Continued.*

257	56715	+	+	+	+	+	+	−	X	X	X	−	−
258	56710	+	+	+	−	−	−	−	XX	XX	−	−	−
259	56711	+	+	+	−	−	−			NVL			
260	56708	+	−	−	−	−	−	X	−	−	−	−	−
261	56719	+	+	−	−	−	−	−	X	X	−	−	−
262	56756	+	+	+	+	+	+	−	X	X	X	−	−
263	56701	+	+	P	−	−	−	−	X	X	X	−	−
264	56753	+	−	−	−	−	−	−	−	X	−	−	−
265	B56671	+	+	+	+	+	−	−	XX	X	X	−	−
266	B56679	+	+	+	−	−	−	−	−	X	−	−	−
267	B56687	+	−	−	−	−	−	−	X	XX	−	−	−
268	B56683	+	+	+	+	+	−	−	X	−	−	−	−
269	B56695	+	+	+	+	+	+	−	X	X	X	−	X
270	B56658	+	+	−	−	−	−	−	X	−	−	−	X
271	B56656	+	−	−	−	−	−	X	−	−	−	−	−
272	B56691	+	+	+	+	+	−	−	XX	X	X	−	−
273	V56681	+	P	−	−	−	−	−	X	X	X	X	−
274	B56694	+	+	−	−	−	−	−	X	−	−	−	−
275	B56673	+	−	−	−	−	−	−	X	−	−	−	X
276	B56657	+	+	−	−	−	−	−	X	X	−	−	−
277	B56677	+	+	+	+	+	+	X	XX	X	−	−	−
278	B40442	−	−	−	−	−	−			NVL			
279	14898	+	+	−	−	−	−	−	X	−	−	−	−
280	B40441	+	−	−	−	−	−	−	X	X	−	−	−

NVL = no visible lesions.

nearly representative of the average condition. A sample of blood was collected from each animal as it was slaughtered and the sample was identified by the number of the reject tag. The postmortem examinations were conducted in the usual manner by Doctors J. E. Sommer, E. J. McLeod, H. S. Koslow and H. Wende. The blood samples and copies of the postmortem reports were returned to this laboratory. The agglutination test used was the technic described by Gilman[4] except that a primary dilution of 1:5.25 was used, giving final dilutions of 1:25, 1:50, 1:100, etc. One page of the record (table I) shows the manner in which the records were kept.

Summary of Results

In all, 336 samples were tested, 87 samples gave a positive reaction to the agglutination test, 237 a negative reaction and 12 a suspicious reaction. About 25.8 per cent of these animals were thus found to be infected with *Br. abortus*.

It is interesting to compare these figures with the results obtained from testing the first 334 blood samples received at this laboratory for diagnosis. These samples came from the southwestern portion of New York State, and all the cattle in this region have passed one or more tuberculin tests. Of this group, 63 gave a positive reaction, 259 a negative reaction, and 12 a suspicious reaction. The rate of infection in this group was found to be 18.8 per cent.

Conclusion

Infection with *Brucella abortus* and tuberculosis in the same individual may be a rather common occurrence in areas where bovine tuberculosis has not as yet been eradicated.

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Iowa in First Place

During 1931, nearly 1,500,000 head of cattle were tested in Iowa, putting the state in first place in bovine disease eradication work. More than 75 per cent of the tuberculin tests were made by 365 local veterinarians. According to the State Department of Agriculture, 675 (89 per cent) of the 725 graduate veterinarians in Iowa are accredited for such work in the state.

TECHNIC OF PHOTOMICROGRAPHY*

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Several years ago, I published a description of a simple, inexpensive photomiocrographic apparatus[1] which was assembled for the most part from various items of discarded laboratory equipment that had been purchased originally for other purposes. Since then I have received many requests for information concerning details of making photomicrographs, and in response to these requests I have prepared this article.

In order to obtain satisfactory photomicrographs of histologic sections they must be prepared to show the exact feature or features which one desires to photograph. The quality of the photomicrographs will be an index of the quality of the sections from which they are obtained. Sections should be thin, but not too thin; a proper thickness is between 7 and 10 microns. They should be well stained so that the various cellular constituents are clearly differentiated. Ordinarily, the hemotoxylin and eosin stain is satisfactory for tissues that have been fixed in formalin solution, although beautiful preparations may be secured by the more elaborate eosin-methylene blue method of Mallory,[2] in which instance the tissues must be fixed in Zenker's solution. It is very important that the sections shall be free from knife scratches or other injurious artefacts. It is inexcusable to exhibit photomicrographs in which knife tears can be seen. One should be thoroughly familiar with the microscopic features which are to be photographed, and much time can be saved by thorough study of the sections before photography is attempted. I have found it convenient to encircle the desired field with India ink applied with a small pen which has a fine point. The ink dries quickly and can be readily removed later by water if desired. The ink is insoluble in xylol, and is not affected if the slide is cleaned with this substance.

The section to be photographed should be placed on the mechanical stage of the microscope, and when the approximate field is found by low-power magnification, the screw controlling the up-and-down movement of the mechanical stage is tightened in order to prevent movement during the exposure. Likewise, it is important that all other adjustable parts are made rigid before

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the plate is exposed. Suitable magnification can be obtained by various combinations of objectives and oculars and by extension of the bellows. Ordinarily an 8x or 10x ocular is used, either of which gives a fairly large flat field so that by extension of the bellows much of the peripheral aberration can be eliminated.

Plates

After trying many varieties of emulsion, I have adopted for practically all exposures a double-coated orthochromatic plate (D-C-Orth-Eastman). This permits much latitude in exposures, and is fairly sensitive to most of the colors encountered in tissue photomicrography when the ray-filter is used.

Exposures

It is in making exposures that most of the difficulty is encountered in the practice of photomicrography. Much experience is required in order to obtain proper exposures under the various color reactions encountered. Even after long experience one is never entirely confident that a given exposure has been the right one. To get some idea of what the proper exposure should be, the trial plate method described by Foot[3] is very useful. By this procedure a plate is placed in the apparatus containing a section to be photographed and the safety cover is removed. After an exposure of about five seconds, the cover is replaced one-fourth the distance and another exposure of five seconds is made. The plate is then covered half the distance and another exposure for five seconds is made. The cover is then replaced three-fourths the distance and a similar exposure is made, after which the plate is covered entirely, removed from the camera, and developed. In this manner the benefit of four exposures on the same plate is secured, and by comparing the various portions, the one which seems to give the best results may be adopted for that particular section. While this is time-consuming, it can be highly recommended to the novice. By making a few trial plates in this manner one soon learns to determine the proper length of exposures by considering the density of the sections and the intensity of the stain as well as the magnification to be used. The higher-power magnifications usually require longer exposures, especially when the dry or the oil-immersion lenses are employed.

At the time the exposures are made, it is important to keep a memorandum of the slide number, the magnification and, perhaps, other pertinent data that will facilitate the identity of the plate after it is developed. Each negative should be

given a number which is recorded on a card containing a descriptive legend of the particular object photographed. These are filed consecutively for future reference. The negative number can be written on the back of the safety cover of the plate-holder with a red wax skin-marking pencil at the time of the exposure, and later transferred in the dark room to the emulsion side of the negative with a soft lead pencil.

Filter

There are several types of photographic filters available, many of which are quite expensive. For all my work I use the diluted Zettnow solution, the formula for which I received from the late Dr. Warthin several years ago. This consists of the following:

Water	250 cc
Copper nitrate	160 gm.
Chromic acid	14 gm.

The mixture is kept in a glass-stoppered bottle, and when used is diluted as follows: Zettnow's solution, 95 cc; distilled water, 140 cc. The solution should be filtered through paper and transferred to a clean, rectangular museum jar, height 5 inches, width 4 inches and depth 0.75 inches. The jar should be fitted with a glass cover and then sealed with suitable adhesive such as marine glue to obviate spilling or evaporation. Other workers may find it essential to have elaborate combinations of filters, but I have found the foregoing satisfactory for all routine exposures.

Developing

For the purpose of developing exposed plates I use the following formula, which is known as "engravers' contrast":

Water	4 liters
Sodium sulphite (E. K. Co.)	192 gm.
Hydroquinon	64 gm.
Potassium bromid	16 gm.
Sodium carbonate	384 gm.

The water should be heated to 125° F. and the ingredients added in the order in which they are listed. Each chemical should be dissolved before the next is added. The solution, which is fairly stable, should be placed in a tightly stoppered bottle. The solution should be used full strength, and developing can be done in a tray or preferably a tank. The solution can be used for a considerable period and fresh developer can be added as the level of the solution recedes. The plates should be raised and lowered several times when they are first placed in the developer

to avoid the possibility of so-called air-cells. When the developer is fresh, the developing time is between five and seven minutes, depending on the density desired. When the developer is older, the time may be extended to as much as fifteen minutes. The solution should never be used hot but should have a temperature between 65° and 70° F. When it becomes blackish brown it must be discarded.

Fixing

After developing, the plates should be rinsed in cold water and placed at once in an acid fixing-bath,* which is made as follows:

Solution A

Sodium thiosulphate (hypo)	960 gm.
Water	4 liters

When thoroughly dissolved the following is added:

Solution B

Water	320 cc
Sodium sulphite (E. K. Co.)	60 gm.
Acetic acid (28 per cent)	192 cc
Powdered potassium alum	60 gm.

(To make 28 per cent acetic acid from glacial acetic acid, dilute three parts of glacial with eight parts of water.)

These chemicals should be dissolved in the order given and care should be taken that the sodium sulphite is completely dissolved before the acetic acid is added. After the sulphite-acid solution has been thoroughly mixed, the potassium alum should be added with constant stirring.

Add solution B to solution A slowly and stir vigorously while mixing. This bath remains clear and fixes clean until exhausted. An attempt to restore an exhausted bath should never be made by adding fresh solution.

The plates should be left in the fixing solution twice as long as it takes the milky color to disappear, after which they should be washed in cold running water for at least fifteen minutes. They should then be gently swabbed on both sides with a generous pellet of wet absorbent cotton, after which they are rinsed and permitted to dry in a room free of dust.

Prints

For making prints I have used the developing paper known as Azo (Eastman). This can be secured in single or double weight in five grades of contrast, varying from that suited for average negatives to that which is adapted for extremely thin or flat negatives. Since many photographic prints are repro-

*Formula from the Eastman Kodak Company.

duced, it is desirable to use a paper with a glossy surface. For the developing of prints I prefer the following formula:

Elon	15 gm.
Sodium sulphite (E. K. Company)	225 gm.
Hydroquinon	60 gm.
Sodium carbonate	315 gm.
Potassium bromid	4 gm.
Water	4 liters

In preparing the solution the water should be 100° F. and each ingredient should be thoroughly dissolved before the next is added. It is advisable to keep this solution in a tightly stoppered bottle and in a dark place where it will keep for several months. For use, portions of the solution are added to equal amounts of water. The mixture should have a temperature of approximately 70° F. when in use.

The exposure necessary in making prints varies with the density of the negative and the amount of light in the printing apparatus. The proper time can be determined by using small strips of sensitive paper and making trial exposures. It is better to obtain a print which develops slowly than one which, through over-exposure, develops rapidly. As each print is made the number of the negative should be written with a soft pencil on the nonsensitized surface. When the print is properly developed it is rinsed in cold water and placed at once in the acid fixing-bath, such as was used to fix the exposed plates. This solution may be used until it begins to turn slightly yellow, or until yellowish stains are noticed on the prints, at which time it should be discarded and an entirely fresh solution obtained. One should never mix used with unused fixing solution in an attempt to rejuvenate it. This solution is inexpensive and the proper fixing of prints and negative requires a solution of dependable strength. After being immersed from ten to fifteen minutes in the fixing-bath, the prints should be placed in cold running water for thirty minutes, and they should be stirred from time to time by the hand or by a wood paddle so as to separate any that may be adhering.

After washing they are ready for ferrotyping, by which process a glossy finish is obtained. The ferrotype tins should be thoroughly polished by applying a small amount of a 10 per cent solution of paraffin (parawax) in xylol. A few drops of this solution are rubbed over the surface of the plate, which is then highly polished with a piece of clean cheesecloth. The prints are placed face downward on the surface of the ferrotype tin and covered with two or three layers of blotting-paper and then

thoroughly rolled with a print-roller. They are then permitted to dry at room temperature.

Recording Prints and Negatives

All exposures should be recorded under a system of negative numbers kept in a filing-case, and the negatives should be filed consecutively in paper envelopes on which the identity number is inscribed. The record cards should contain, in addition to the negative number, pertinent data relative to each exposure, such as the case number, the salient features delineated, and the magnification. It is convenient to file copies of prints from each negative in an album beneath each of which is inscribed a legend descriptive of the particular picture. By this means it is possible at any time to find prints which may be desired with a minimal expenditure of time.

For a more technical description of photomicrography the paper by Petrunkevitch[4] should be consulted. For those desiring information concerning the essentials of photography, "The Fundamentals of Photography,"* by C. E. K. Mees, is recommended.

*Obtainable from the Eastman Kodak Company.

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Dog Bites and Rabies in New York City

In an effort to reduce the number of dog bites occurring in New York City, the Health Department has made constant efforts to rid the city of stray animals. Presumably as a consequence, during the past few years the incidence of rabies has decreased considerably, but the number of cases of dog bites remains around 14,000 annually. It was thought that stray dogs were the chief offenders in the matter, but an analysis of dog bites during the first four months of 1932 showed that, of the 64 per cent occurring in streets and other public places, 48 per cent were by dogs known to have owners, and only 16 per cent by stray dogs. This indicates that the chief offender is the dog whose careless owner allows him to be at large.

STUDIES IN INFECTIOUS ENTERITIS OF SWINE
VI. Immunity in Swine Coccidiosis*

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Subsequent to the completion of earlier investigations on swine coccidiosis, steps were taken to study the histopathology of this condition. It was observed then that when sporulated swine coccidia were fed to pigs, an infection resulted which followed a characteristic course. After the administration of the strains used at that time, newly-formed oöcysts began to appear in the feces six or seven days later. The elimination of newly-formed oöcysts usually continued for periods of from ten to fifteen days, after which time the infection cleared up if the pigs were kept on clean floors and separate from others to which cultures were being fed. Six pigs were each fed coccidia and subsequently killed at varying intervals between the fourth and fourteenth days following the administration of the infective forms, for the purpose of studying in detail the various developmental stages of the parasite while in the tissues, together with its effect upon the intestine.

The coccidia that were used in this series were obtained from a different source than those used during the original studies. The small intestine is invaded by those fed in this project. Of a large number of histologic slides made from sections taken at regular intervals from the intestines of pigs killed for this purpose, only a few showed, after careful examination, the presence of a very small number of intracellular forms. In view of the enormous number of sporulated coccidia which were fed, the development of a definite resistance on the part of the host was suggested. Our attention was called to the results of the fecal examinations made on this and other groups. The fecal examinations made on the pigs killed prior to the sixth or seventh day after the administration of infective forms were negative (this interval being the duration of the prepatent period), and therefore were of no aid.

On the basis of our previous data, fecal examinations made on pigs that were allowed to live eight to fourteen days following experimental feeding of sporulated oöcysts should show numer-

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ous newly-formed oöcysts in the feces. The records of these pigs showed the elimination of only a small number of oöcysts as determined by the sugar concentration method. The small number of oöcysts passed could not be attributed to a subnormal condition of the culture used because it was young and showed no degenerated forms and a portion which was used on other pigs for building up additional culture material produced heavy infections, as was indicated by the elimination of large numbers of newly-formed oöcysts.

Our records show also that when the same group of swine was fed sporulated coccidia for the purpose of increasing the amount of culture, the first infection produced an enormous number of oöcysts, while the second and third infections showed a smaller number and when the third feeding of culture followed closely the preceding ones the resulting infections were extremely light. The group infected for the purpose of studying the histopathology during infection had been given sporulated forms *per os* daily until destroyed. These data and literature references on the question of coccidial immunity in general prompted us to direct some attention to the problem in swine. The comparatively long sporulation time of Eimeria from swine and the definite course and self-limiting nature of the infection observed make swine a favorable species in which to study this problem.

Literature Review

The literature indicates some differences of opinion regarding the development of immunity to coccidia and the mechanism involved. Crawley[1] concluded that immunity does not develop following infections with coccidia and that the host can be reinfected after an attack. Wetzel[2] described coccidiosis in the cat as a definitely self-limiting disease. He did not admit the production of immunity by infections but stated that repeated reinfections may produce a chronic form of the disease. Young[3] reported that chickens given three feedings of sporulated oöcysts following an interval of one month did not become immune. In his project the infection followed a limited course. Although Klimmer[4] observed that older animals may harbor the infection without manifesting visible symptoms, he stated that immunity is not present following the subsidence of an attack.

Conversely, other workers support the conception of the development of some form of resistance or immunity following coccidial infection. Greater susceptibility of the young to coccidiosis is frequently recorded, although no data are given re-

garding possible previous infections in the older animals. Fantham[5] and Jowett[6] both observed that the disease runs a milder course in older fowls. Reich[7] reported that practically all the rabbits which he autopsied in Berlin showed evidence of intestinal or hepatic coccidiosis without manifesting symptoms, demonstrating in his opinion that rabbits are able to resist light infections. Because of the marked resistance to coccidiosis possessed by rabbits in later life, he stated that possibly they even acquire a certain degree of immunity.

Hall and Wigdor[8] recorded the self-limiting character of Isospora infections in the dog. In one of their experimentally infected dogs they were unable to produce reinfections by giving three successive feedings of sporulated Isospora. They suggest the production of an immunity as a result of the first infection. Velu,[9] discussing coccidiosis of the goat in Morocco, stated that it appears exclusively as an affection of the young. Wasielewski[10] asserted that the cause of the transition from the acute form to a chronic form in the rabbit remains to be determined. Regarding cattle he noted that older animals appeared to be only slightly affected but that an absolute immunity did not exist. However, he raised the question whether the decreased susceptibility of the older calves is not based upon their recovery from coccidial infections during early life. Beach and Corl[11] fed 33 chicks infective coccidial oöcysts a second time without mortality or sickness while 34 per cent of a control group not previously infected died from coccidiosis. They indicated that possibly the previously infected chicks had acquired a resistance for coccidia.

Andrews,[12] working with dogs and cats, presented proof of the production of immunity to Isospora infections. He found that these hosts were not immediately susceptible to reinfection and stated: "This immunity lasts for seven months, and probably for life." Johnson[13] found that in the chicken a definite immunity is produced by infections. His data show that resistance is not dependent upon the age factor alone, that when birds were reared without exposure to coccidiosis they were still highly susceptible regardless of age. Johnson also noted that one or two inoculations of small numbers of oöcysts did not result in much protection against a second feeding of oöcysts. However, the administration of a large number of oöcysts produced considerable resistance to subsequent feedings.

Yakimoff and Galouzo[14] observed, in Russian localities where bovine coccidiosis is endemic, that chiefly the calves developed

the disease, while both young and old became sick in localities where the disease was said to have appeared for the first time. This manifested resistance in the older animals they interpreted as evidence of immunity attributed to previous infections. Chapman[15] studied the disease in an isolated rabbit colony and concluded that a light infection may produce a slight degree of relative immunity. Tyzzer[16] furnished substantial proof of the development of immunity in the common fowl in response to infections with coccidia. He found also that the species which invade the intestinal wall of the host produce a more marked protection in a shorter time than those coccidia which do not invade the deeper tissues of the host.

Bachman[17] concluded that coccidial infections in the rabbit result in the development of some immunity to the subsequent ingestion of infective oöcysts and that age itself is not a factor in the appearance of immunity.

METHODS

The same strains of oöcysts were used throughout this study dealing with the development of immunity. At no time were new coccidia introduced from the field. The cultures used to produce experimental infections consisted of forms simulating *Eimeria scabra* and some resembling *Eimeria debliecki*, as described by Henry.[18] One of these proposed species will be treated in detail in a separate report.

The large amount of infective material required for this project was obtained by feeding sporulated oöcysts to susceptible swine. This resulted in a great multiplication, with the formation of oöcysts, which were eliminated in the feces. Such feces were mixed with water and passed through a series of copper sieves to remove the larger particles. Sufficient 10 per cent potassium dichromate solution was added to give the suspension a content of from 2 to 2½ per cent potassium dichromate, depending upon the viscosity. (During the early stage of the infection, considerable mucus and serum are usually present in the excreta.) The mixture was then kept in liter serum bottles and aerated at least once each day. Sporulation usually occurred in from 10 to 15 days, depending upon the amount of aeration to which the suspension was subjected. Before feeding the sporulated oöcysts, the dichromate was removed by dialysis in parchment tubing.

The severity of the infections produced was measured by the number of oöcysts found in the feces. Composite fecal samples

from the individual pigs were collected from the pen-floors each day and placed in quart mason jars. Sufficient water was added to permit the suspension to be mixed thoroughly and filtered through a fine copper screen. To one part of fecal suspension were added two parts of concentrated sugar solution. The contents of the tubes were mixed and centrifuged at not less than 1,800 r.p.m. for three or more minutes. The oöcysts were harvested from the surface by means of a blunt glass rod, placed on a slide and examined under a cover-slip. Cover-slips of the same size were used throughout the project. If large numbers of oöcysts were present, the average of ten high-power fields was determined and converted to a low-power reading. When the number was not large the average was ascertained directly from 10 low-power fields. It must be emphasized that the number of oöcysts recorded should not be considered in the sense of being specific numerical counts but rather as representing the degree of infection. When a large number of determinations is made on the same animals, it gives a very satisfactory picture of the course and severity of the infection.

Interval Exposures

When susceptible pigs were fed infective forms on one or two successive days, the elimination of newly-formed oöcysts commenced, following a prepatent period of about seven days. The elimination continued for about fourteen days, after which the feces became free of oöcysts, provided the pigs were not subjected to additional infections. The oöcyst-elimination curves following one or two feedings of infective forms on successive days are shown in charts 1a, 1b and 2.

It will be seen that one or two experimental feedings at different intervals produced varying degrees of resistance. However, an absolute immunity to subsequent infections (using the same species) did not result. Three susceptible pigs (3441, 3442, 3443, chart 1a) were given one dose of sporulated oöcysts *per os.* The approximate number of oöcysts eliminated by each pig was determined in accordance with the previously described method. The course and degree of infection is indicated by a composite plotting in chart 1a. In response to the first inoculation the reaction was quite pronounced, the number of newly formed oöcysts found being as high as 937 per low-power field on one day. Twenty-seven days subsequent to the disappearance of the oöcysts from the feces, twice the amount of sporulated oöcysts used to produce the first infection was fed to the same pigs.

The reaction was comparatively mild in contrast to that of the first infection. The increased amount of infective material was used in the second inoculation to make certain that a diminished reaction could not be attributed to a decreased number of infective oöcysts. This reaction is typical of other trials not described in this report.

Pigs 3862, 3863, 3864, 3865 and 3866 (chart 2) were fed in a similar manner except that the interval between the first and second infective feeding was shorter than in the first series (chart 1a). A diminished number of oöcysts likewise appeared in response to the second infective dose (chart 2). In this group the first infection, however, was not so severe as in the first two groups of pigs used. This probably is due to a low-grade natural infection suffered by this group during its stay in the feeding-lots previous to being placed in the experiment pens. The third inoculation of oöcysts followed 40 days after the close of the second infection and resulted in a greater elimination than in the second infection, which had followed more closely upon the first, suggesting that the degree of the acquired immunity decreased with the lapse of time.

Similar results were obtained in other groups. Four check pigs (3870, 3871, 3872 and 3873, chart 2) not previously infected experimentally were used in connection with the third infection. These were fed the same amount of culture as pigs 3862, 3863, 3864, 3865 and 3866. A more severe infection resulted, the oöcyst elimination being almost three times as great, indicating a much greater susceptibility. Fifteen days after the termination of the third infection these two groups, together with two additional check pigs (3874 and 3875) not previously infected, were given two infective doses each on successive days.

The severity of the infection, as determined by the elimination of oöcysts, was materially reduced in the previously infected groups, especially in pigs 3862, 3863, 3864, 3865 and 3866, which eliminated only a few oöcysts. The last check pigs (3874 and 3875) passed large numbers of oöcysts. When these same groups were again infected on the 150th and 151st days of the project (22 days following recovery from the previous infections), the two groups of check pigs showed a slightly increased resistance, while the group infected for the fifth time showed an elimination slightly higher than it manifested in the previous infection, although this slight apparent increase might be attributed to factors associated with the collection of samples, such

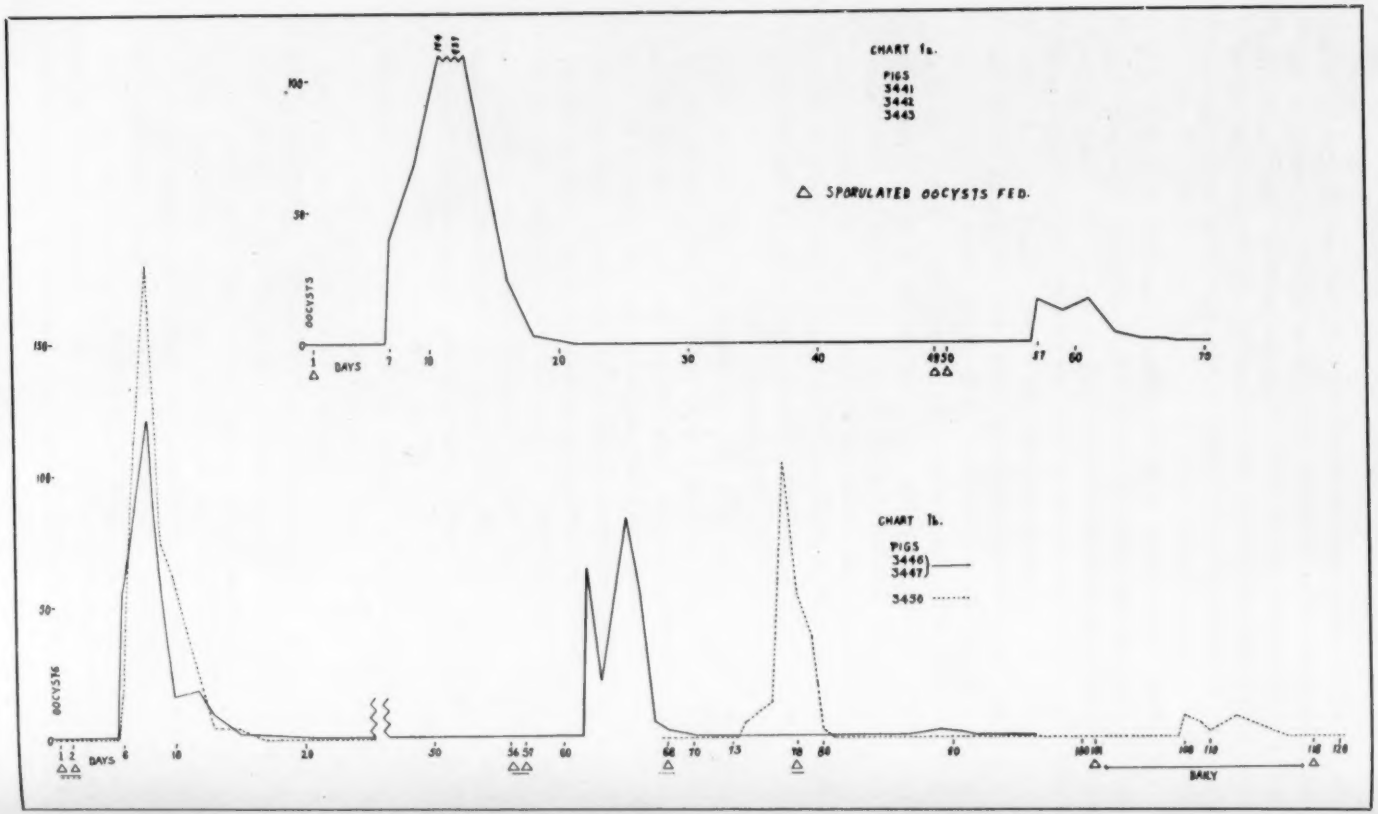


FIG. 1. Charts 1a and 1b, showing results following feeding of sporulated oöcysts.

as the bulk of the feces passed at the time or inherent deviations of the method of determining the elimination.

The reinfection of pigs at different intervals was carried out somewhat differently with three pigs (chart 1b). They were each given oöcysts on two successive days. The oöcyst eliminations of pigs 3446 and 3447 are expressed by a composite plotting, while that of pig 3450 is carried separately. The first infection resulted in a typical course of oöcyst-elimination in each pig. Pigs 3446 and 3447 were again fed 40 days subsequent to recovery. Pig 3450 was fed 12 days later from the same culture series.

The response in each case was quite similar, although the number of oöcysts eliminated during the second infection was not decreased so strikingly as in some series. The number of days that elapsed between these infections (40 and 52 days) must be considered. A portion of the same culture administered to pig 3450, on the 68th day of the experiment, was fed to pigs 3446 and 3447 on the 78th day of the project. The later feeding, following closely upon the second infection, produced the third infection in pigs 3446 and 3447. The oöcyst elimination was almost negligible in this case, indicating that the pigs were almost immune at the time of the third inoculation which followed closely upon the second attack. By this order of infecting the pigs in this experiment, the second infection of pig 3450 served as a check against the culture which was given as a third infective dose to pigs 3446 and 3447. Pig 3450 (chart 1b) also was infected a third time, to ascertain the degree of immunity developed as a result of the second infection. In this case infective oöcysts were given daily from the 101st to the 118th day, and the results show that the amount of exposure is not capable of influencing appreciably the extent of the infection under a given body resistance. The third infection of pig 3450 was strikingly less severe when compared with its second infection.

Repeated infections after short intervals produced an increased resistance to coccidiosis. This acquired resistance, however, is not permanent in the pig. It gradually diminishes with the lapse of time following each infection. This fact is brought out more clearly in subsequent experiments. The curves of oöcyst elimination also support the view that coccidiosis in the pig is a self-limiting disease. Other individual pigs and groups reacted to infections in the same manner as those shown in the charts. It also establishes the fact that an absolute immunity

FIG. 1. Charts 1a and 1b, showing results following feeding of sporulated oöcysts.

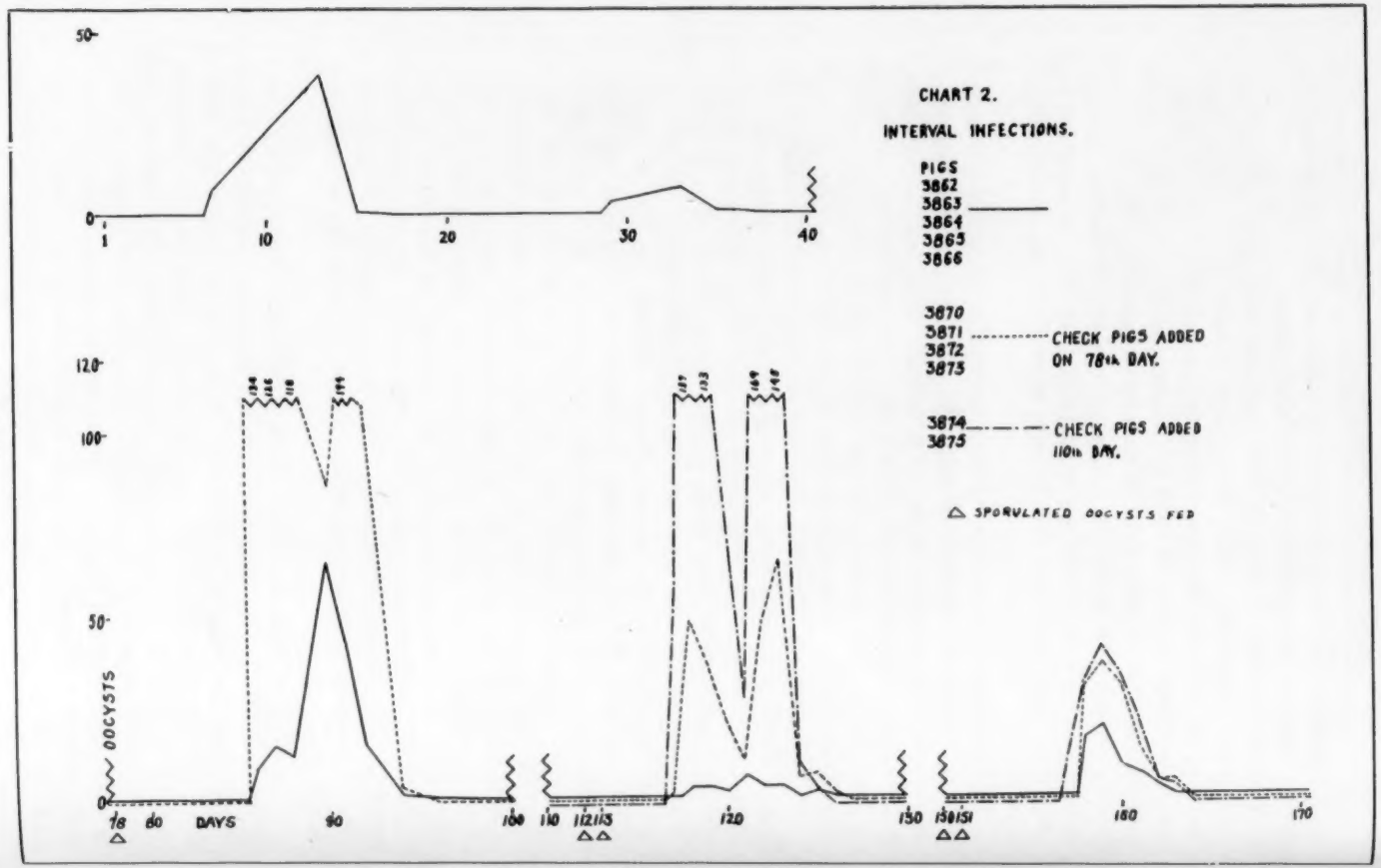


FIG. 2. Chart 2, showing results of feeding at intervals.

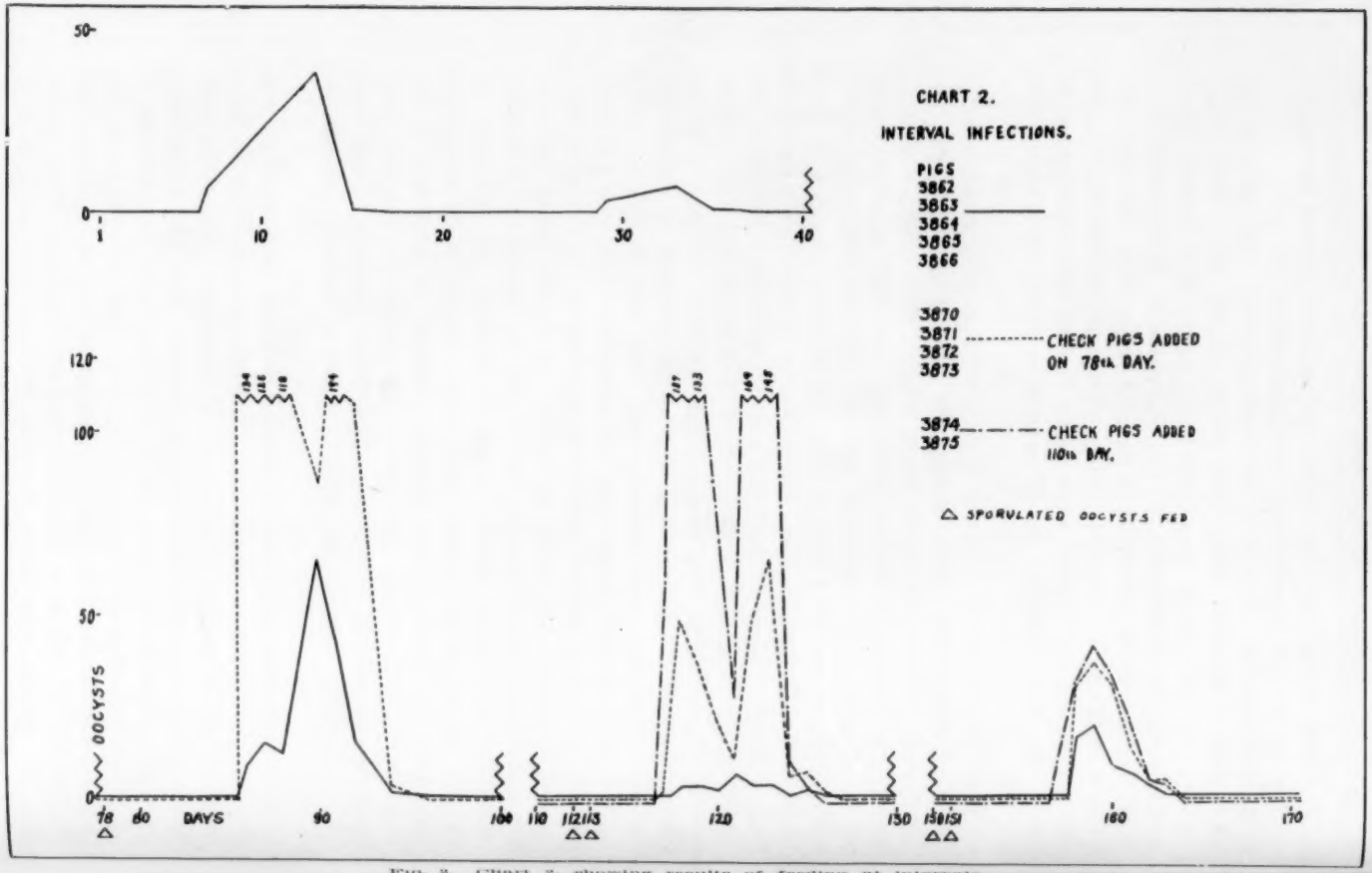
is not produced in the pig by one or two heavy inoculations on consecutive days. Spontaneous recovery also takes place in pigs which are naturally infected, provided they do not ingest additional infective forms.

In every case when pigs were infected, their feces became free from oöcysts after a period of about 21 days subsequent to the ingestion of sporulated forms, provided no further infective forms were ingested. In two instances pigs continued the elimination of very small numbers of oöcysts for a longer period. These were subjects used to increase our supply of oöcysts and they were kept in the same barn and attended by the same attendant who cared for the series of pigs which were fed sporulated oöcysts daily over a long period of time.

After noting the development of varying degrees of resistance to coccidial infections following the ingestion of sporulated oöcysts, an attempt was made to render swine completely immune. With this objective ten boars and gilts weighing about 90 pounds were given infective coccidial oöcysts daily in the feed over a long period (chart 3a). Five of this number (pigs 3755, 3756, 3719, 3757 and 3758) each received daily 25 cc of culture *per os*, while five others (pigs 3747, 3748, 3749, 3750 and 3751) each received 200 cc daily. The different amounts of coccidial suspensions were given in order to ascertain the effect, if any, that greater dosages might have upon the rapidity of immunization.

The plottings in chart 3a represent the courses of the infection in these two groups and are based upon a composite of the individual daily determinations made on each pig. The daily feedings of oöcysts were continued in all pigs until the 79th day. The elimination of oöcysts became quite light after about 30 days and continued at a low ebb until the 51st day, notwithstanding the daily administration of infective forms. Following a short period when no oöcysts were passed, a slight elimination occurred subsequent to the 56th day. After this the feces continued negative, although the daily administration of infective forms continued. Pig 3752, whose reaction is shown from the 35th to the 61st day, served as a check. This pig was fed part of the same serial numbers of culture given to the pigs shown in chart 3a.

The check pig was fed at a time when the two groups in chart 3a were almost immune, *i. e.*, were passing only small numbers of oöcysts, although large numbers of infective forms were given daily. However, a pronounced response, as is evidenced by the



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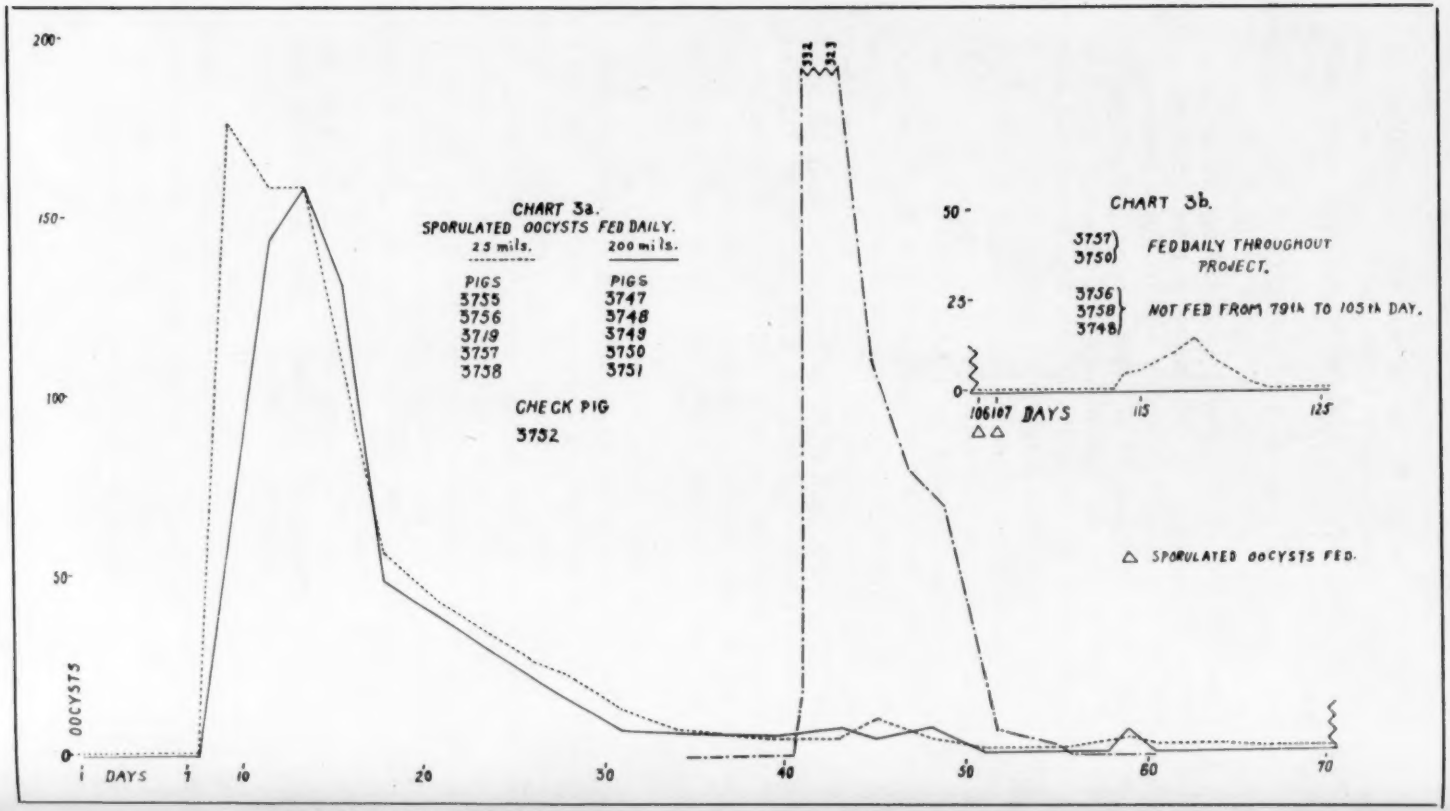

Fig. 3. Charts 3a and 3b, showing results of daily feedings throughout experiment.

elimination of newly-formed oöcysts, resulted in the check pig, indicating that a definite resistance had developed in the group inoculated daily, which was not due to the lack of viability of the coccidial suspension fed.

The oöcyst production in the group receiving 25 cc daily was very similar to that of the group receiving 200 cc of suspension. The slight differences which appear undoubtedly can be attributed to individual variations in the pigs and to some extent to the method used in determining the oöcyst elimination.

The daily feeding of sporulated coccidia was continued beyond the 79th day in pigs 3757 and 3750 (chart 3b). Pig 3757 continued to receive daily 25 cc of coccidial suspensions until the 125th day, while pig 3750 continued to receive 200 cc daily. A break is shown between charts 3a and 3b, from the 70th to the 105th day. During this time neither of the pigs fed infective coccidia daily showed the presence of newly-formed oöcysts in the feces. Pigs 3756, 3758 and 3748 received no sporulated forms from the 80th to the 105th day, but on the 106th and 107th days of the experiment were given *per os* 200 cc of sporulated oöcysts. Pigs 3757 and 3750 each likewise received at the same time a similar amount of the same oöcyst suspension. The pigs (3756, 3758 and 3748) which had been given a rest period of only 26 days following immunization did not thereafter possess the same high degree of immunity which they manifested previous to the rest period, as was evidenced by the elimination of newly-formed oöcysts in response to the ingestion of infective forms on the 106th and 107th days. However, the pigs that received feedings daily throughout the period of the experiment were not susceptible, since no newly-formed oöcysts were found in their excreta by the sugar centrifuge concentration method.

A second experiment, using six pigs, was instituted for the purpose of confirming the results obtained on the development of a complete immunity produced by daily inoculations of coccidia and determining in greater detail more about the duration of such resistance in the absence of exposure to infection. The results are shown in chart 4. Pigs 3459 and 3460 were fed infective forms every day during the entire course of the experiment, which continued for 210 days. The remaining four pigs (3455, 3456, 3457 and 3458) were given infective forms daily only during the first 100 days. Before the end of 100 days, all pigs ceased to eliminate oöcysts, even though infective forms were being fed daily at the time, indicating the establishment of complete immunity.

FIG. 3. Charts 3a and 3b, showing results of daily feedings throughout experiment.

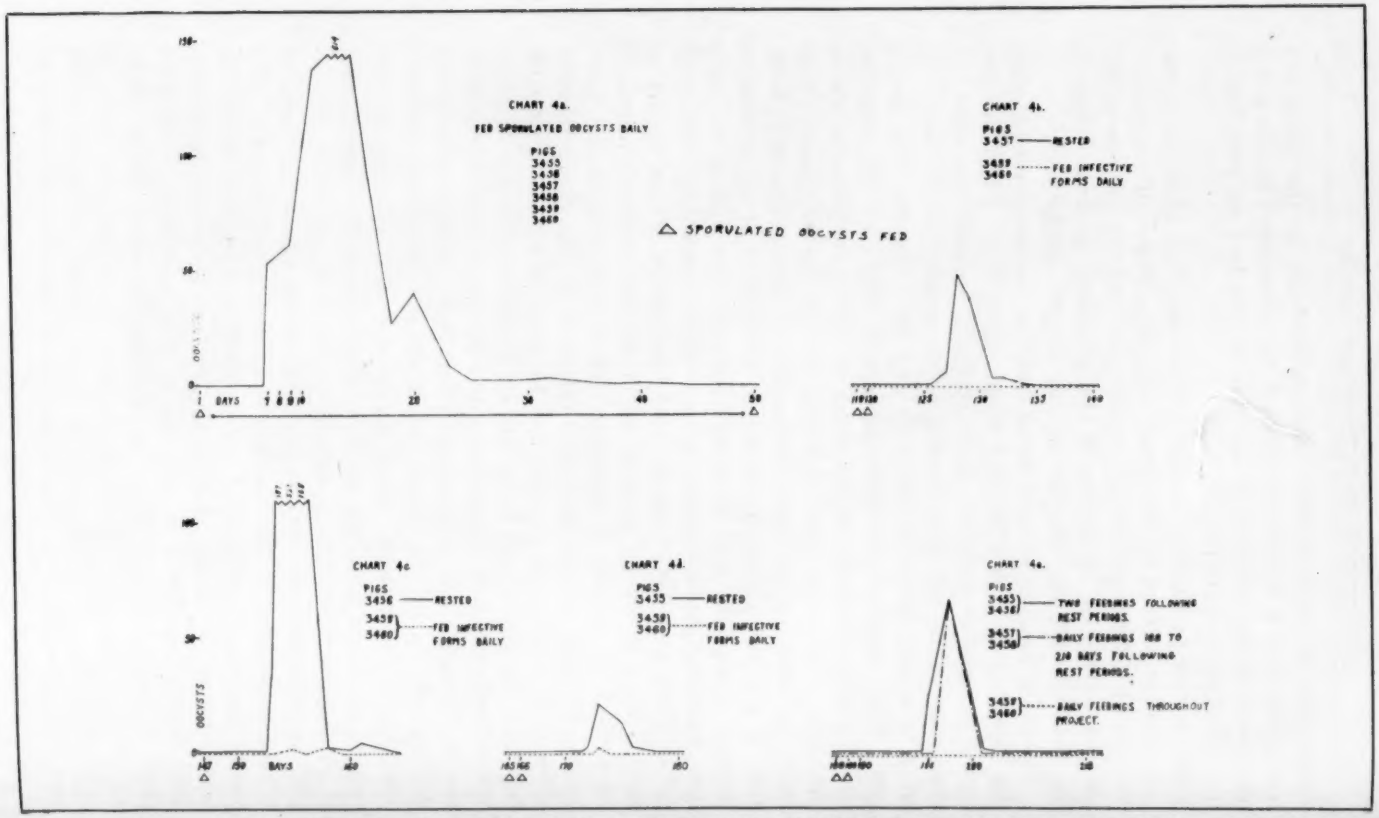

FIG. 4. Charts 4a, 4b, 4c, 4d and 4e, showing results of daily feedings following rest period.

On the 119th and 120th days, large quantities of sporulated oöcysts were given to pig 3457, which had been rested 18 days and also to the control pigs 3459 and 3460. No infection resulted in the latter two, while the rested animal (pig 3457) proved again susceptible, as is shown by the elimination of oöcysts (chart 4b). On the 147th day, pig 3456, which had been rested for 49 days, was given a large amount of sporulated culture. Pigs 3459 and 3460, which were fed daily, were each given a similar amount. A very marked infection took place in the pig which had been rested 49 days, while the pigs fed daily eliminated only a few isolated oöcysts on several days (chart 4c).

On the 165th and 166th days, pig 3455, which had not been infected since the 100th day of the experiment, and the daily-fed check pigs were each given *per os* a large quantity of suspension of sporulated oöcysts (chart 4d). In the check pigs a barely perceptible infection occurred, as was indicated by the small number of oöcysts passed. Pig 3455 gave a greater response, although the infection was not so severe as in the previously fed pigs that had been rested. An individual variation has been observed in some members of other groups. In this connection it should be mentioned that pig 3455 was the last one of its group to discontinue the elimination of oöcysts during immunization. What, if any, relation this fact may have to the course of subsequent infections is not known.

On the 188th and the 189th days, pigs 3459 and 3460 were given two doses of infective oöcyst suspension many times greater than those received previously. Fecal examinations proved them to be immune. A similar amount of culture was fed on the same days to pigs 3455 and 3456, each of which had been infected once during a rest period subsequent to previous immunization (charts 4c, 4d and 4e). A moderate infection resulted, as is shown by the composite plotting. Pigs 3457 and 3458, both infected once during a rest period following immunization, were given infective oöcysts daily from the 188th to 210th day (chart 4e). The curve of elimination was almost identical with that of pigs 3455 and 3456, fed only twice. The reactions of pigs 3457 and 3458 are unlike the reactions obtained when fresh susceptible pigs are infected daily for the first time, in which case the oöcyst elimination is prolonged slightly beyond that obtained by feeding one or two doses of infective forms on successive days. In the cases of 3457 and 3458, it appears that only a limited susceptibility existed, which was promptly changed to a state of complete immunity. Histologic studies of the intestines of swine infected during various stages of

FIG. 4. Charts 4a, 4b, 4c, 4d and 4e, showing results of daily feedings following rest period.

immunization to be reported later are of interest in this connection.

Chart 5 shows the composite oöcyst elimination of 8 pigs given one or, in some instances, two feedings of infective oöcysts on successive days and that of 16 other pigs fed sporulated oöcysts daily.

It will be noted that the groups given sporulated oöcysts only once or twice on successive days recovered in about 21 days following the ingestion of infective forms. The cessation of oöcyst elimination indicated the termination of the infection in these

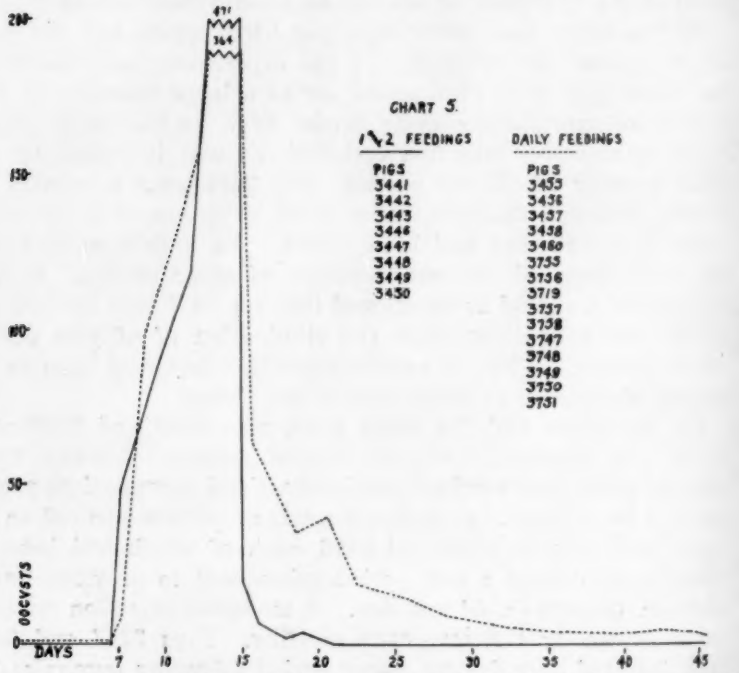

FIG. 5. Chart 5, showing results of single and double feedings compared with daily feedings.

pigs. This was the result of the completion of that part of the cycle of the parasite taking place in the body cells rather than the result of an absolute immunity on the part of the host. It was ascertained that swine could be reinfected following recovery from infections produced by feeding infective oöcysts on one or two successive days, no matter how large the dose given. On the other hand, the pigs given infective forms daily continued the elimination of oöcysts until the 48th day in one group and somewhat later in another (chart 5), at which time

the host, by means of some defensive mechanism, had developed sufficient resistance to withstand the further ingestion of infective forms. This shows that a state of susceptibility, although somewhat decreased, still prevailed in the group fed only once or twice.

In every group of pigs naturally or experimentally infected, the disease proved self-limiting, provided the pigs did not receive additional infective forms. The pig is a favorable animal in which to study this phase of the infection because of the comparatively long time required for swine coccidia to sporulate. The only cases of prolonged infections observed by us in swine, comparable to the so-called chronic infections cited by some workers, occurred in pigs which were kept in the same barn and fed by the same attendant who cared for a group of pigs that were given daily feedings of sporulated oöcysts. The pigs which were fed infective oöcysts daily spilled feed containing such forms or carried quantities of contaminated feed from the trough to the floor on their feet. It was necessary in this barn for the attendant to enter each pen daily, which no doubt accounts for the transfer of infection from the pigs fed sporulated coccidia to the unfed pigs. These, however, were the only instances encountered which resembled so-called chronic infections. When the pigs which were fed daily were moved to another barn and attended by a different caretaker, the continued low-grade infections terminated in those which were not being experimentally infected.

Our studies indicate clearly that the asexual cycle is limited and that prolonged infections (sometimes called chronic infections) are due to reinoculation. This reinfection is almost unavoidable in some species, *i. e.*, where the parasite has a short period of sporulation and the hosts have hair-coats or feathers not readily cleansed (rabbits, sheep and chickens) permitting the parasite to adhere to the covering of the host and there undergo further development. Man is a favorable host for the study of this problem. In the case of man, Dobell and O'Connor[19] observed that when humans infected with coccidia were held under observation, the oöcysts soon disappeared from the feces.

While working with avian coccidiosis, we found it practically impossible to avoid reinfection in the chicken. Sporulation of coccidia from the common fowl can take place in from 21 to 48 hours, depending upon the species. It is possible for droppings to adhere to the feathers near the vent and sporulate before reaching the ground. After experimentally infecting chickens,

we sometimes found sporulated oöcysts on the floor of the cage, which was renewed every 24 hours and the specimens examined two hours after collection. Even under such conditions reinfections are possible.

SUMMARY

Experimental coccidial infections in swine followed a definite course, resulting in a characteristic curve of oöcyst elimination after a prepatent period of about seven days.

One experimental feeding produced varying degrees of partial resistance to later infections but did not result in an absolute immunity, nor was it possible to produce an absolute immunity by feeding massive doses of sporulated oöcysts on one or two successive days.

The administration of infective forms following recovery from previous infections caused by one or two feedings resulted in the production of increased resistance to subsequent infections, provided the intervals between infections were of short duration.

Such partial immunity, acquired as a result of an infection, decreased with the lapse of time.

A complete immunity was produced by the daily administration of infective forms. The immunity produced in this manner was not permanent. Following rest periods of 18 days or longer, it was again possible to infect those pigs which had been fully immune previous to the rest period.

Following one administration of coccidia to pigs not previously infected heavily, the elimination ceases about 21 days later. Such pigs are still susceptible, although they have acquired a partial resistance as is shown by lighter subsequent infections. This conclusion is further supported by the fact that when swine are given infective forms daily, they will continue the elimination of oöcysts for a much longer time, indicating that some degree of susceptibility extended beyond the 21st day. It also suggests that the cessation of oöcyst elimination in those given only one infective dose must be due to the completion of the coccidial development in the host and not to a host immunity.

Coccidiosis in swine is a self-limiting infection. The only instances of prolonged oöcyst elimination simulating so-called chronic infections were due to continued light reinfections and promptly disappeared when exposure to such infections ended.

One group of 5 pigs given eight times the amount of culture fed to a similar group did not show any appreciable difference in the rate of the development of immunity.

Varying degrees of immunity of the host may retard the time of the first appearance of oöcysts. However, the smaller number of oöcysts passed might account for these results, *i. e.*, the number of oöcysts passed during the first stage of elimination may be so small as to be missed by even the concentration method. The duration of oöcyst elimination is somewhat affected by the degree of resistance possessed by the host. Highly susceptible pigs may eliminate oöcysts for fourteen days or more, as determined by the sugar concentration method (chart 5). Infections following shortly upon previous ones may be characterized by periods of elimination as brief as five days (chart 4d).

The appearance of oöcysts in the feces following the administration of infective forms was sometimes delayed 24 to 48 hours in pigs previously infected. This is illustrated in the last two series of infections plotted on chart 2.

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Canada had two well-known members at Atlanta: Drs. A. E. Cameron, of Ottawa, and J. A. Campbell, of Toronto.

MASTITIS IN EWES, CAUSED BY INFECTION WITH A PASTEURELLA*

By HADLEIGH MARSH, *Bozeman, Montana*
Montana Veterinary Research Laboratory

Every summer sheep-owners have reported cases of "blue bag" in ewes on the range in August and September. This condition is apparently distinct from the udder inflammations which occur during the lambing season, and it is difficult to understand why such a disease should appear in ewes on the range when the lambs are three or four months old. Sheep-owners report as high as 5 per cent of the band affected. There is some mortality and the ewes that recover have permanently "spoiled bags."

During the past summer, we had opportunity to make observations on two outbreaks of this type of mastitis. In both instances we found infection with a specific organism, the same bacterium being present as the causative agent in both bands of sheep.

The first outbreak which we investigated occurred in a band of 1,200 ewes which were being summered on a dry range consisting partly of grass and partly of old wheat land, on which there was a growth of Russian thistle. The sheep were seen by us on August 7. The herder reported that ten ewes had died with mastitis between August 1 and August 7. At this time we saw six cases of the disease, one of which was very weak and was killed for autopsy.

On August 11, the whole band was worked through a chute, and all ewes showing any sign of mastitis were cut out. Nineteen were cut out of the band of 1,200, and were taken to the ranch headquarters. The herder was instructed to keep the band away from old bed-grounds and to camp only one night in a place.

On August 18, the band was again seen, and the herder reported only four more cases occurring after cutting out the affected ewes on August 11. At this time no cases of mastitis were found in the band.

The second outbreak from which we obtained material occurred in a band of 800 ewes during the latter part of September, after the sheep had been trailed from the summer range in the mountains to the ranch. Several of these sheep died. On September 30, a ewe which had first shown symptoms on September 29 was brought to the laboratory for study. In this

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case the symptoms, lesions, and bacteriological findings were identical with those observed in the first band of ewes.

Symptoms

The disease apparently develops very rapidly, producing an acute systemic disturbance with a high fever and loss of appetite. After the first 48 hours the fever subsides, but the local process in the udder continues to develop. The ewe may die within a week, probably from a toxemia, or the udder breaks down forming a chronic abscess, and the ewe returns to its normal condition except for the local lesion.

Temperatures were taken on 17 sheep. Five of these cases showed high temperatures, ranging from 105° to 106° F. These

Fig. 1. Mastitis case from first outbreak described. Right mammary glands much enlarged.

were all apparently in the early stages of the disease. In three of these cases the temperature was back to normal within three days. The twelve other sheep showed normal temperatures at the time they were observed. Most of these were evidently in the later stages of the disease.

During the acute stage of the disease, there is loss of appetite. In all cases observed, only one side of the udder was affected, being greatly enlarged and hard. The milk-cistern contains a yellow or grey liquid material containing white flakes. Later, in the fatal cases, there is a blue discoloration of the tense skin over the greatly enlarged gland.

In the cases which do not terminate fatally, abscess formation takes place after about a week, with necrosis of the gland tissue, and eventual opening of the abscess and sloughing of practically all of the gland tissue. This process may continue for three months. In these cases the ewe quickly returns to normal general condition after the first week.

HISTOPATHOLOGY

Sections of the diseased gland showed all capillaries engorged with blood, and hemorrhages into many of the acini. Epithelium of the acini was destroyed, although a few intact nuclei remained. In the acini and between them, were masses of broken-down chromatin material in granules of varying size, and degenerated amorphous cytoplasmic material.

ETIOLOGY

With the purpose of determining whether this type of mastitis is a specific infection, a bacteriological study was made of the two outbreaks. Cultures were made from the secretion of the affected mammary gland of three ewes in the first outfit and one in the second, and in all cases an almost pure culture of a Gram-negative bacillus was obtained. All strains of this bacillus from the four ewes were studied in detail and were found to be identical in cultural reactions. This organism is a Pasteurella, and corresponds in its reaction to Jones' Type 3 *P. boviseptica*,[1] except that our organism ferments raffinose. The cultural reactions of the organism are as follows:

Morphology: Smears from exudate show a very small cocco-bacillus, varying to a rod of some length. Smears from serum-agar slant show small rods varying in length from 0.8μ to 2.7μ. Width about 0.5μ.

Staining: Gram-negative. Bipolar. Longer forms show tendency to irregular staining.

Motility: Non-motile.

Agar slant: Growth moderate, diffuse, smooth, showing slight bluish tint in young cultures by transmitted light.

Gelatin: Not liquefied.

Blood-agar: No hemolysis.

Indol: Not produced.

Nitrates: Reduced in four days.

Hydrogen sulfid: Produced in three days.

Litmus milk: Acid, no coagulation.

Carbohydrate reactions: No gas in any sugar. Acid produced in glucose, lactose, sucrose, xylose, mannitol, glycerol, maltose, sorbitol, galactose, raffinose and levulose. No acid in dulcitol, dextrin, inulin, salicin, mannose and arabinose. The fermentation of lactose is consistently slight and does not show for three days. The fermentation of glycerol appeared at three days.

Pathogenicity: Two strains killed guinea pigs, and two strains failed to kill. Intraperitoneal injections were used.

The predisposing factors in this infection are unknown. It seems probable that bruising of the udder by the lamb is a factor, as sixty-pound lambs are often very rough in their nursing. This would explain the occurrence of mastitis late in the summer. The transmission of the infection in the band probably occurs on bed-grounds, when the sheep are camped repeatedly at the same place.

FIG. 2. Mastitis case from second outbreak described. Acute stage. Right mammary gland, much enlarged.

EXPERIMENTAL CASE

A culture of the organism was inoculated into the udder of a healthy lactating ewe.

August 12: 5:00 p. m. The growth of one agar slant was taken up in 3 cc of saline solution, and 2 cc of this suspension was inoculated into the milk-duct of the right teat.

August 13: 8:00 a. m. Temperature 106.7° F. Respiration 64. Right mammary gland full and hot. Milk apparently normal. 11:15 a. m. Temperature 106.3° F. Right side of udder enlarged and becoming hard. Milk appeared normal, but very little could be milked out. 5:00 p. m. Temperature 106.5° F. Given 50 cc of a 1 per cent acriflavine solution intravenously.

August 14: 8:00 a. m. Temperature 104.3° F. Respiration 26. Ewe depressed. Some flakes in milk. 5:30 p. m. Temperature 103.8° F. Milk thinner and like whey.

August 15: 8:00 a. m. Temperature 103.2° F. Secretion from udder thin and colorless. Physical condition of udder unchanged.

August 17: The ewe died. The udder was removed and the pathological condition was found to be exactly similar to that of the field case. The specific Gram-negative bacillus was recovered.

TREATMENT

In view of the fact that intravenous injections of acriflavine have been found to be of value in mastitis of cows, this treatment was tried on the ewes. Thirteen ewes in the first band investigated were given intravenous injections of 50 cc of a 1 per cent solution of acriflavine. The sheep treated were selected

FIG. 3. Same case as shown in figure 2.

as apparently not advanced cases. One of these sheep was treated a second time after four days. The treatment failed to check the inflammatory process in the udder, as in all cases abscess formation and sloughing followed. Some of the treated ewes died.

A single attempt was made to test the effect of acriflavine in the case of the experimentally infected ewe. Eighteen hours after inoculation, a milk sample was taken and a bacterial count

made. Six hours later, 50 cc of acriflavine solution was given intravenously. Eighteen hours after the acriflavine treatment, the milk showed a slight yellow color and another bacterial count was made. The bacterial count was much higher than before treatment. The inflammatory process continued and the ewe died. In this case the acriflavine intravenously was ineffective.

Any treatment is difficult to apply under range conditions, because the mastitis develops so rapidly. The preventive measures were based on the assumption that the infection was spread on the bed-grounds. All visibly infected ewes were removed from the band, and the herder was instructed to camp at a new place every night. Apparently these measures were effective in controlling further spread of the disease.

Comparison with Mastitis of Sheep as Described in Europe

Infectious mastitis of ewes has been investigated in Europe, and there are many reports in the literature on this disease. Miessner and Schoop[2] give the results of their investigation of mastitis in sheep, and give a brief review of the literature. Their description of the symptoms and lesions corresponds very closely with our observations.

Miessner and Schoop consider the bacillus of Dammann and Freese[3] as the causative organism, and call it *Bacterium mastitidis*. The cultural reactions as described by them are somewhat indefinite, and do not correspond exactly to the characteristics of the organism which we find. However, it seems possible that we are dealing with the same organism. This possibility is supported by the fact that they find their *Bact. mastitidis* causing pneumonia in lambs, which indicates that their organism may be a Pasteurella.

Miessner and Schoop report good results from immunization of ewes with a bacterin prepared from the *Bact. mastitidis*.

Summary

Mastitis occurs quite frequently in range sheep in August and September, when the lambs are three or four months old.

It is characterized by a sudden onset with high fever, and acute inflammation of one side of the udder. In some cases death results, while in the majority of cases the udder abscesses, and the ewe recovers, but the udder is permanently spoiled.

This type of mastitis is a specific infectious disease caused by a Pasteurella.

No successful treatment has been developed.

Successful immunization with a bacterin has been reported in Germany.

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Business Fellowships to Agricultural Students

Students from each of thirty agricultural colleges in the United States and Canada were recently awarded business fellowships by the Danforth Foundation and Purina Mills. The awards comprise four weeks' special training in commercial and agricultural leadership. Wm. H. Danforth, founder of the Danforth Fellowship Plan, which is now in its fourth year, feels that this special summer training gives the students the opportunity of contacting business at close range during their college course, so that after graduation they may more quickly find the work for which they are best fitted.

Two weeks of the training is devoted to a study of the manufacturing, sales promotion, and research methods of a large feed-milling organization at Saint Louis, Mo. Studies in farm management on the Purina Experimental Farm, Gray's Summit, Mo., and trips to grain exchanges and stock yards are a part of the plan. Following this, two weeks are spent at the American Youth Foundation Camp, near Shelby, Mich.

It is the custom for the Dean of Agriculture in each of the colleges represented to select three candidates for the award. One from each college is then chosen by officials of the Foundation.

Alabamans at Atlanta

An even score of Alabama veterinarians registered: Drs. B. Z. Burleson and D. J. Meador, Montgomery; C. A. Cary, M. W. Emmel, I. M. Hays, I. S. McAdory, Houston Odom, R. S. Sugg and E. S. Winters, Auburn; Clive Daly, J. B. Favara and D. A. Piatt, Birmingham; A. R. Gissendanner, Dothan; T. B. Howle, Oxford; J. D. Ratchford, LaFayette; J. H. Ryland, Camden; W. D. Staples, Anniston; C. Stewart, Cullman; J. C. Webb, Piedmont; E. E. Williams, Wetumpka.

Make a note of the dates for the Chicago meeting next year—August 14-15-16-17-18, 1933.

PARATUBERCULOUS ENTERITIS IN SHEEP CAUSED BY AN ACID-FAST ORGANISM*

By J. A. HOWARTH, *Davis, California*
Division of Veterinary Science, University of California

A disease of sheep, resembling Johne's disease in cattle, has been observed in a band consisting of 2,000 head, in the Sacramento Valley of California. The clinical manifestations of this ailment are a general emaciation, unthriftiness, tucked-up abdomen, intermittent diarrhea, and occasionally an elevation of temperature. There is a marked reduction in the milk secretion; the appetite does not seem to be greatly impaired.

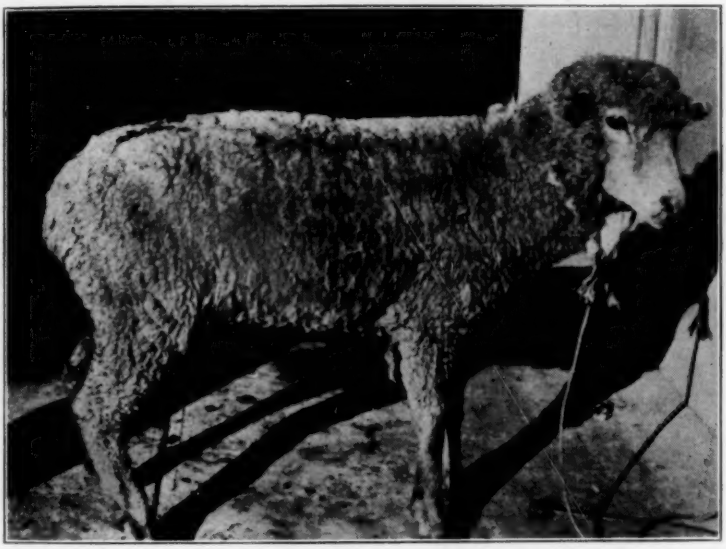

FIG. 1. Ewe 8, which reacted to avian tuberculin. Note characteristic tucked-up abdomen, emaciation and unthriftiness. Autopsy revealed characteristic lesions. Acid-fast organisms also demonstrated.

Sheep affected with this disease survive for long periods; some improve at times, only to have relapses later. The affection is confined mostly to aged sheep. During gestation the symptoms and lesions seem to be intensified; mortality is always greater after lambing-time. This disease could easily be confused with a heavy parasitic infestation, especially when observed in the field. On the ranch where the disease has appeared, the loss has

*Received for publication, March 28, 1932

been approximately 20 to 30 head each year for the past seven years. This estimate includes only sheep showing symptoms similar to those here described, for all unthrifty and emaciated animals were repeatedly treated for parasites, whereas this group did not respond to such medication.

On postmortem examination the carcass is usually emaciated, the intestinal tract being the seat of the greatest involvement. There is a great thickening of the walls of the large and small intestines and, in the more advanced cases, of the cecum. The mucous membrane is greatly thickened, corrugated, and thrown up in irregular folds. In many cases the mucous membrane is studded with hemorrhages. There is a serous infiltration not only of the outer coat of the intestinal tract but also of the mesenteric

FIG. 2. Section of large intestine showing the mucous membrane greatly thickened, corrugated, and thrown up in irregular folds.

attachment, the mesenteric lymph-glands being enlarged, grayish in color, and succulent.

Smears, made from deep scrapings of the affected parts of the intestinal mucosa and stained by the alcoholic acid-fast method, showed great clusters of small, acid-fast bacilli. Smears made in the same manner from a great number of the mesenteric lymph-glands also showed acid-fast bacilli.

Deep scrapings of affected tissues from the large and small intestines were made into an emulsion with normal saline, and antiformin was added to the extent of 20 per cent. The material

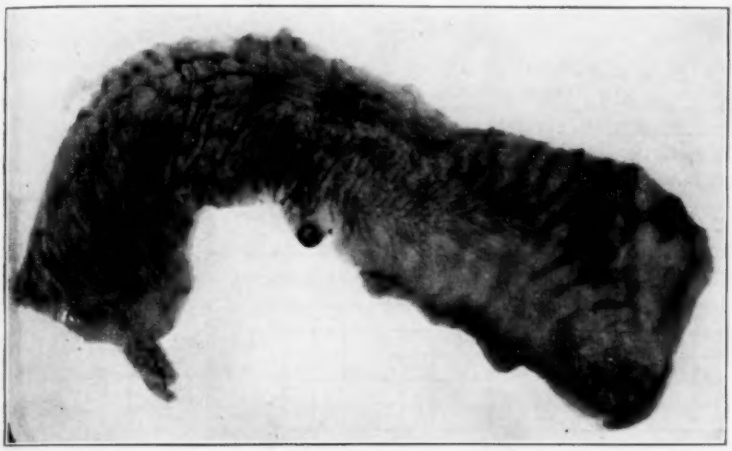

FIG. 3. The wall of the cecum greatly thickened, thrown up in irregular folds, and studded with dark hemorrhagic areas. The dark, round nodule at the lower center margin is the ileo-cecal valve, which is markedly hemorrhagic.

was then placed in the shaking-machine for one hour. At the end of this time it was centrifuged and the supernatant fluid drawn off. Normal saline was then added to the residue, well mixed, and again centrifuged. After the material had been washed in

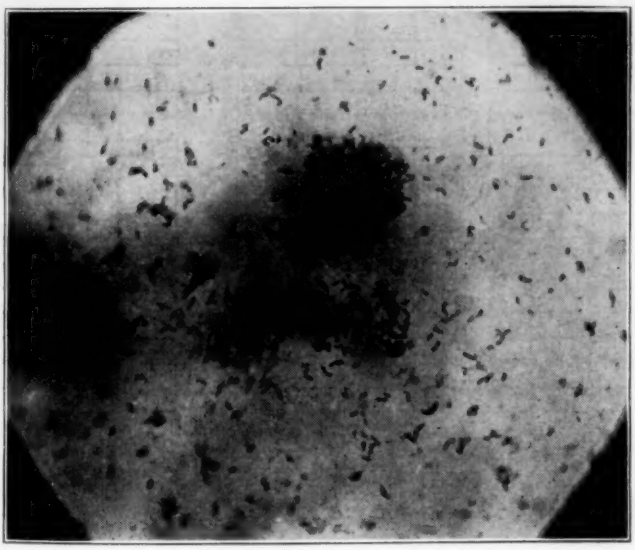

FIG. 4. Acid-fast organisms in smear from submucosa of small intestine.

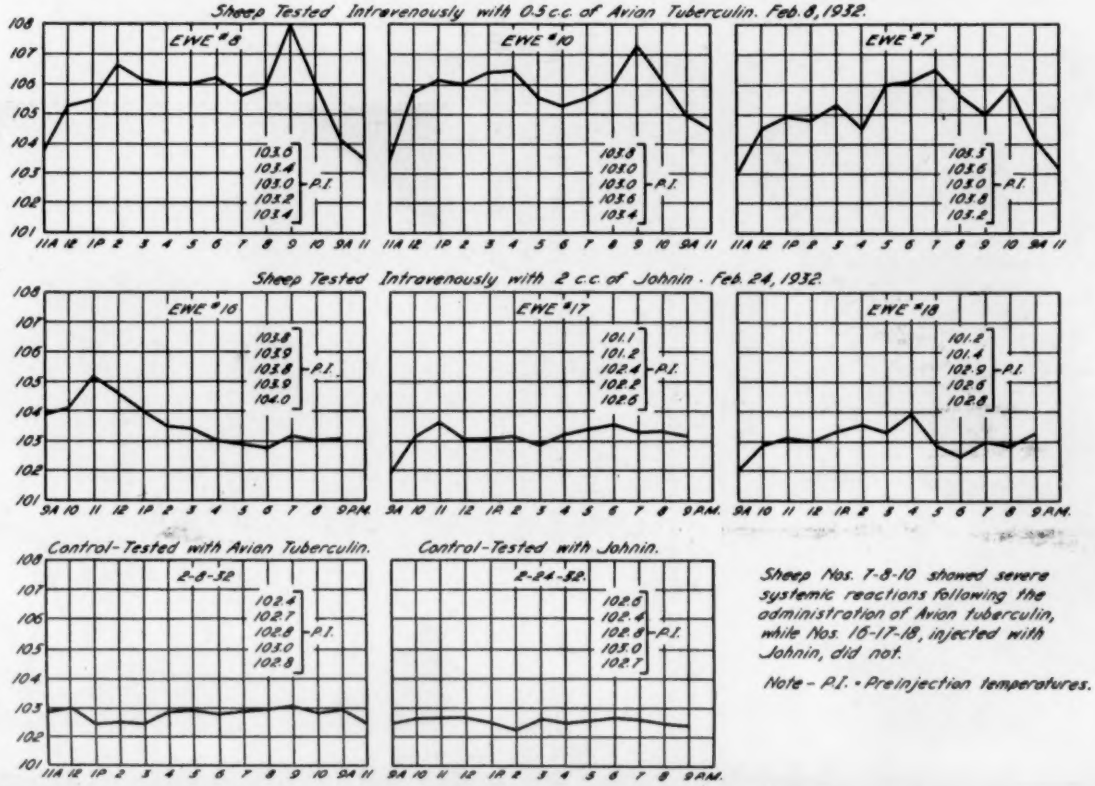

FIG. 5. Temperature charts of sheep tested with Johnin and avian tuberculin.

this manner three times the residue from the final centrifuging was used as the inoculum. The latter was then seeded on Twort and Ingram's[1] basal glycerol-egg medium, in which 1 per cent bovine tuberculin was substituted for the added nutrient. With an incubation temperature of 37.5° C., approximately four weeks elapsed before growth was observed.

As this affection was believed to be Johne's disease, many of the animals were tested with johnin and avian tuberculin. Affected sheep gave a better reaction to avian tuberculin than to johnin. These animals, after an intravenous administration of 0.5 cc of avian tuberculin, exhibited a severe systemic reaction and a rise in temperature of 3 degrees (F.) or more. Some sheep tested intravenously with 2 cc of johnin showed no systemic reaction; others, only a slight one. The sheep which gave the thermal reaction to the johnin test, although it did not give a uniform rise in temperature, showed characteristic lesions, and acid-fast organisms were demonstrated in the smears taken from the affected parts. There is a possibility that affected sheep would show a more severe reaction to johnin if larger doses were administered. This procedure will be undertaken at a later date.

The symptoms, the lesions, and the finding of acid-fast organisms indicate that this affection may be Johne's disease, and further investigations are now being carried on.

Although Johne's disease has been reported in sheep in Great Britain by Stockman[2] and by M'Fadyean, Sheather and Edwards,[3] the writer is unable to find any published record of the occurrence of this disease in sheep in North America.

Fig. 5. Temperature charts of sheep tested with johnin and avian tuberculin.

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Radio Broadcasts at Atlanta

A feature of the convention was a series of radio broadcasts over Station WSB, on top of the Biltmore Hotel, arranged by Dr. L. A. Mosher:

Tuesday, Aug. 23—"The Southern Veterinarian and Our Live Stock Industry," by Dr. N. F. Williams, Fort Worth, Tex.

Wednesday, Aug. 24.—"Veterinary Medicine and Human Health," by Dr. R. R. Dykstra, Manhattan, Kan.

Thursday, Aug. 25—"Live Stock Health and Wealth," by Dr. John R. Mohler, Washington, D. C.

DEVELOPMENT OF THE RIGHT OVIDUCT IN DOMESTIC FOWL*

By TH. C. FITZGERALD *and* FRITZ VOLKMAR
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In a group of nine Black Giant hens, purchased for use in the course of topographical anatomy of domestic animals, two of these hens revealed, during dissection, the presence of a persistent right oviduct. Following is a brief presentation of the findings:

Fowl 312: Breed, Black Giant; sex, female; age, over one year; condition, fat; weight, 8¼ lbs.; weight of the liver, 91 gm (2.43 per cent of the body weight). The single ovary is in the usual anatomical position. The ova range in size from 2 to 25 mm in diameter. The oviduct to the left of the median plane is 30 cm in length. The oviduct to the right of the median plane is 38 cm long. The width of the latter ranges from 10 mm at the isthmus to 35 mm at the vagina. The right oviduct opens into the cloaca lateral to the rectum, the left oviduct enters dorsal to the rectum. The density and size of the folds of the mucosa of the right oviduct are very similar to those of the left.

Fowl 324: Breed, Black Giant; sex, female; age, over one year; condition, fat; weight, 8 lbs. 7 ounces; weight of the liver, 100 gm (2.6 per cent of the body weight). The single ovary weighs 15 gm and is in the normal anatomical position. The ova are not well developed. Their diameters average 2 to 3 mm in size. The oviduct to the left of the median plane is 45 cm long. The oviduct to the right of the median plane is likewise 45 cm in length. The width of the latter ranges from 12 mm in diameter at the isthmus to 35 mm in diameter at the vagina. The entrances of the oviducts into the cloaca were corresponding in position. The right duct entered the right lateral wall of the cloaca and the left duct entered the left lateral wall. The density and size of the folds of the mucosa were very similar in both ducts.

The average weight of the seven other hens was 2.8 kgm. The average weight of their livers was 64 gm (2.7 per cent of the body weight).

*Received for publication, April 2, 1932.

From the physical appearance of these persistent right oviducts and from the fact that a free communication to the cloaca existed, it is evident that they were capable of functioning. Persistent right oviducts, capable of functioning, seem to be extremely rare occurrences in domestic fowl. A search of the literature revealed only three previously reported cases.

Gallagher[1] observed, in an actively laying hen, a right oviduct which was nearly as long as the left oviduct and equally voluminous. Its anterior extremity exhibited an infundibular membrane in contact with the ovary. Posteriorly it opened into the cloaca opposite the left oviduct. The appearance of its mucous membrane suggested that it was capable of functioning. Kaupp[2] shows the photograph of the genital organs of a hen which had developed two ovaries and two oviducts, stating that both oviducts were functioning. Veenendaal[3] reproduces the illustration of a fowl-situs exhibiting two well-developed and functioning oviducts.

Cases of rudimentary persistent right oviducts, characterized by *atresia tubae caudalis*, and of cystic right oviducts are more frequently reported in literature. It is not unlikely that they may be more common than is assumed at present. Their repeated finding in fowl is pointed out in the current text-books on veterinary anatomy, dealing with poultry. Mention is made in them of earlier occurrences. Of the more recent observations, the following may be briefly called to memory.

Dsenit and Undritz[4] describe nine cases of rudimentary and cystic right oviducts. They were connected with the cloaca by fibrous ligaments. Andres[5] describes nine cases of rudimentary right oviducts encountered in 2,000 autopsies. These oviducts ranged in length from 2.5 to 36 cm. Connection with the cloaca was either by contact or by ligament. Curson[6] describes and illustrates a persistent right oviduct which was two-thirds the length of the left one. Both oviducts were closed toward the cloaca. He also mentions and illustrates two cases of right cystic oviducts. McKenney[7] encountered two cases of persistent right oviducts among 450 poultry autopsies. In one of the hens the right oviduct seemed capable of functioning, since part of six yolks were found in its lumen. Continuity of the right oviduct could not be established. Reinus[8] studies the pathogenesis of the cysts of the right oviduct of fowls. He observed that the wall of the cysts has great similarity to the mucosa of the oviduct and he concluded that failure of certain parts of the Mül-

lerian duct to regress causes congestion of its secretions, leading to cyst formation. These cysts continue to increase in size with the growth of the fowl.

With the finding of an interesting anomaly the alluring question as to its origin always arises. In pursuance of this thought it was profitable to review certain phases of chick embryology.

The development of both oviducts is said to begin uniformly from a cranial and a caudal part. The cranial part originates on the fourth or fifth day from the Müllerian duct. The origin of the one of the left side is more cranial than the one of the right side. At about the fifth or sixth day of incubation, differentiation of the sexes begins to appear. At this point of development the oviducts are of equal size and length. Subsequent to sex-differentiation, the right ovary and the right oviduct regress and the left oviduct alone continues to grow.

There has been much speculation as to the reason for the regressive development. The inquiry into the reason for the regression of the right oviduct will unquestionably find its solution in the discovery of the influences which arrest the development of the right ovary. In the light of the newer knowledge of the growth-regulating properties of hormones, it is feasible that under normal conditions the left ovary exerts an inhibitory influence upon the right ovary and oviduct. Disturbance or destruction of this influence would naturally cause the right ovary and oviduct to develop and to function.

Inquiring into the possible causes for such a disturbance of the growth-regulating influence, one is met by the well-known fact that malformations are caused by heat-disturbance during incubation. An explanation on the basis that injury from a severe change of temperature during incubation would result in an irregularity of the mechanism regulating the growth development of the right oviduct should not meet with any serious objection.

Since the right oviduct was capable of functioning and has probably functioned, the further question arises, whether there was a compensatory equalization in the growth and development of the left oviduct or not. The average length of the left oviduct in normal fowl is said to be 80 cm. Measurements of the oviducts in the remaining seven Black Giant hens, which could have furnished a basis for the comparison, have not been taken. From the facts established in the present investigation it is evident that no correlation exists between the liver weights in fowl having a single oviduct and in fowl having paired oviducts. It

is not known whether the development of the right oviduct adversely affects the size of other organs.

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Visitors at the JOURNAL Office

During the past two months quite a number of members have visited the new A. V. M. A. headquarters in Chicago. The latter part of June, Dr. T. A. Sigler, of Greencastle, Ind., dropped in, between sessions of the Democratic Convention being held a few blocks away. The next was Dr. W. J. Embree, chief veterinarian of the Western Weighing and Inspection Bureau. Dr. Herman Busman called just before leaving Chicago to take up his new B. A. I. duties in Indianapolis. Several days later, two other B. A. I. veterinarians, Dr. L. Enos Day, of Chicago, and Dr. H. B. Raffensperger, of Moultrie, Ga., dropped in for a short visit.

Dr. J. E. Shillinger, of the U. S. Bureau of Biological Survey, Washington, D. C., called to talk over the work of the Committee on Agricultural Extension Service. Dr. L. A. Merillat stopped just long enough to pick up some French veterinary journals which he is abstracting for the JOURNAL. Dr. T. H. Ferguson, of Lake Geneva, Wis., came in to talk over a number of A. V. M. A. matters to come up at Atlanta. Dr. J. T. Hernsheim, of Chicago, came to inspect the new offices and get information on transportation to Atlanta. Drs. Richard F. Eagle and A. A. Swaim, of Wilson and Company, Chicago, called to pay their respects and incidentally consult the A. V. M. A. library. Dr. A. E. Behnke, of Milwaukee, Wis., had just enough time between trains, returning from a trip in the East, to take a run out to the office and say "how d'y'do." Alexander Eger, known to practically every veterinarian in America, was among those who accepted the invitation to call and look us over. He wanted to talk books, of course.

Quite a list for two months.

LEUCOCYTE COUNTS ON THE BLOOD OF NORMAL, CHOLERA-INFECTED AND RECENTLY IMMUNIZED PIGS*

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The purpose of this investigation was to study the effect of the simultaneous treatment on the white blood cells of pigs, and to obtain more data on the white cell count of normal and cholera-infected pigs.

Dinwiddie,[1] in 1914, reported white cell counts of normal and cholera-infected hog blood. From seven normal pigs he obtained counts as low as 7,000 per cubic millimeter and placed the average at approximately 11,000 per cmm. Corresponding blood counts made on fourteen cholera-infected pigs at different stages of the disease, varying from the first to the twentieth day after infection, revealed a distinct leucopenia from the beginning, which even in the earliest stages was usually so marked as to be discernible in stained smears.

Lewis and Shope[2] have stated that hog cholera appears to be the only acute infectious disease of swine that is characterized by a decrease in the number of white blood cells, and consider the leucopenia of hog cholera to be so pronounced as to be of diagnostic importance. They found the white cell count in normal pigs to vary from 14,000 to 24,000 per cmm.

Cahill[3] found the leucocyte count of normal susceptible pigs to vary from 10,300 to 16,000 per cmm. and places the average at 13,000.

The foregoing citations, which by no means exhaust the literature on the subject, are presented for the purpose of showing: first, that the leucopenia of hog cholera is a well recognized phenomenon, and, secondly, that the white cell count of normal hog blood has been found to vary in a considerable range.

EXPERIMENTAL WORK

In most cases the blood samples were examined immediately after they were drawn, without the addition of anti-coagulants. The blood was obtained either by pricking the vein on the dorsal surface of the ear or by clipping off the end of the tail. It was collected in a small pool on a glass slide, drawn immediately into the mixing pipette, the diluting fluid added and the sample

*Received for publication, April 4, 1932.

shaken. One-half per cent acetic acid was used as diluent. The usual equipment for making blood counts was used and counts were made in the usual manner. In addition to the white cell counts made from fresh blood samples, many samples were collected in the field. In these cases, the practice suggested by Lewis and Shope[2] was followed, that is, the blood was drawn from the tail into graduated tubes or bottles containing 20 milligrams of powdered potassium oxalate per 10 cc of blood, the dilutions being prepared at the laboratory and counts made in the usual manner.

In making counts daily from the same pig, it became necessary to take precautions against drawing blood from an area inflamed from previous trauma, experience having shown that an exaggerated count is thereby obtained. It has therefore been our practice to draw successive samples from the right ear, the left ear and the tail, in turn, and then to repeat the cycle in case no inflammation is apparent.

White Cell Counts of Normal Pigs

One hundred and thirty-one counts were made on 48 apparently normal pigs. Considerable variation was noted in individual pigs on different days and some pigs quite regularly showed higher counts than others. Variations of from 12,000 to 39,000 per cmm. were found in this group of pigs, or an average of approximately 20,000 per cmm. In most of the experiments described in this paper, no particular time of day was selected for making the leucocyte counts. Results of work done later in the investigation, however, indicate that more uniform counts could be obtained if they were made at the same time each day, particularly with reference to the time of feeding.

Most investigators have found that there is a wide variation in the number of leucocytes in the blood of normal pigs, but few data are available as to the causes for such variation. In an attempt to throw some light on this subject, the following experiment was conducted.

Twenty-five counts were made on eight normal pigs before feeding and 25 counts were made on the same pigs after feeding. Of the 25 counts made after feeding, 23 showed an increase, one remained the same and one showed a decrease. The average of the 25 counts made before feeding was 17,742 and the average of the 25 counts made after feeding was 22,893. There was thus an average increase after feeding of 5,151, or 29 per cent. The counts before feeding were made in the morning, approximately 17 hours after the preceding feed. The ones after

feeding were made approximately 1¾ hours after the morning feed.

The pigs in this experiment were fed a moderate amount of a well-balanced ration. No attempt was made to determine the effects on the leucocytes of various kinds or amounts of feed. A study of this kind, however, might reveal additional interesting information. Table I shows the results of counts made before and after feeding.

TABLE I—*Leucocyte counts of normal pigs before and after feeding.*

PIG	WEIGHT (LBS.)	DATE (1931)	COUNTS*		INCREASE OR DECREASE
			BEFORE FEEDING	AFTER FEEDING	
1	45	4–18	13,750	23,906	+10,156
		4–20	20,781	25,468	+ 4,687
		4–21	20,937	26,250	+ 5,313
		4–22	20,781	24,218	+ 3,437
2	45	4–18	16,406	16,875	+ 469
		4–20	22,812	20,937	− 1,875
		4–21	18,750	23,437	+ 4,687
		4–22	17,187	17,968	+ 781
3	45	4–18	15,750	22,187	+ 6,437
		4–20	17,031	25,937	+ 8,906
		4–21	18,593	25,000	+ 6,407
		4–22	16,250	25,937	+ 9,687
4	45	4–18	14,375	16,875	+ 2,500
		4–20	16,562	19,375	+ 2,813
		4–21	18,437	22,500	+ 4,063
		4–22	15,000	17,187	+ 2,187
5	40	4–23	16,875	16,875	
		4–24	15,000	17,187	+ 2,187
		4–25	12,968	20,156	+ 7,188
6	40	4–23	27,500	39,843	+12,343
		4–24	19,062	22,968	+ 3,906
7	40	4–23	18,903	32,187	+13,284
		4–24	14,687	21,875	+ 7,188
		4–25	20,937	22,656	+ 1,719
8	45	4–23	14,218	24,531	+10,313

*Counts were made in the morning approximately 17 hours after the preceding feed, and again approximately 1¾ hours after the morning feed.

WHITE CELL COUNTS OF VIRUS PIGS

Sixty-five counts were made on fifteen pigs which were used to furnish simultaneous and hypering virus. The pigs in this

group were off feed by the fifth day and in some instances as early as the third day. They were killed for virus from the sixth to the tenth day, inclusive, and on autopsy showed lesions of uncomplicated hog cholera. In the case of some of these pigs, counts were made daily throughout the course of the disease or until the pig was killed, but in most instances a different group of pigs was counted on different days. From the history, clinical symptoms and postmortem lesions of the pigs in this group, it was considered that the virus was of high virulence and subsequent tests showed such to be the case. The results of the white cell counts of these pigs are given in table II. A study of this table shows that leucopenia is not always well marked, even in what was considered unquestionable cases of acute hog cholera.

In addition to the counts on pigs used to furnish virus, 140 counts were made on pigs in the various stages of cholera. These additional counts were made on cholera-infected pigs which were not suitable for virus production, pigs brought in for diagnosis and cases of natural infection on farms. The counts in this group were in general agreement with those of the virus pigs.

White Cell Counts Following Serum-Alone Treatment

In this experiment, as well as the ones concerned with white cell counts following the simultaneous treatment, the pigs were placed in clean disinfected pens. White cell counts were made once a day for three days prior to treatment and for several days following. Only two pigs were used in this experiment and as no reduction in the number of leucocytes was apparent, it was not repeated.

White Cell Counts Following Simultaneous Treatment

Lewis and Shope[2] state that "the administration of virus and hyperimmune serum, the so-called double treatment, to hogs does not produce a leucopenia, so that the leucocyte count is available for use in detecting the cause of trouble in vaccination breaks." Cahill,[3] on the other hand, found the leucocyte count in two simultaneously treated pigs to be reduced the day following treatment to 7,300 and 4,400, respectively. After 24 hours, the decrease ceased and a gradual leucocytosis occurred.

In the present experiments, counts were made on three successive days before treatment and the average of the three counts was taken as a basis for comparison. The pigs were then in-

TABLE II—*Leucocyte counts of virus pigs.**

DAYS AFTER INJECTION	1	2	3	4	5	6	7	8	9	10
PIGS COUNTED	6	8	3	10	7	10	10	5	3	3
Leucocyte counts	19,000 16,000 15,000 12,500 31,875 21,000	7,500 14,218 17,343 11,250 6,250 15,000 13,750 12,800	11,500 16,400 12,800	6,406 29,531 5,156 14,218 12,500 10,000 6,400 14,000 10,000 5,600	6,000 12,000 5,000 14,000 9,200 9,000 5,100	9,218 23,750 12,187 5,000 11,700 5,000 4,000 17,500 11,000 4,300	6,093 9,687 8,593 9,000 10,000 3,300 5,000 13,000 11,800 5,900	4,843 5,468 10,000 5,000 4,000	5,312 5,000 10,000	5,600 6,000 9,600
Averages	19,229	12,225	13,566	11,381	8,614	10,365	8,137	5,862	6,937	7,066

*These counts were made on a group of 15 pigs, which ranged in weight from 40 to 90 pounds and were considered suitable for virus production.

jected with varying doses of anti-hog cholera serum and 2 cc of hog cholera virus. Counts were made daily for several days after treatment.

Four pigs were used in the first experiment and were given an ordinary field dose of anti-hog cholera serum. By comparing the counts made after treatment with the average of the three counts made before treatment, it will be seen that the four pigs injected with serum and virus all showed a drop in leucocytes following the treatment. This drop was apparent on the third day after treatment and the return to normal took place within two to four days following the initial drop. The leucopenia was most marked in the case of pig 102, which showed a well-marked drop beginning the third day and continuing until the sixth day. The detailed record of counts for pigs in this group is given in experiment 1 (table III).

As the results of the preceding experiment seemed to be somewhat different from the work reported by Lewis and Shope,[2] it was decided to obtain more data on the subject. In the second experiment, the pigs were all given the simultaneous treatment, but the dose of serum was varied somewhat. Three pigs were given the regular dose of serum as recommended by serum-producers, and three were given approximately seven-tenths of said amount. Like the previous experiment, this one tends to show that there is a definite drop in the number of leucocytes following the simultaneous treatment. In general, it appeared that the drop in leucocytes was greater in the low-dose pigs, both as to minimum count and the duration of reaction. Detailed record of the counts for the pigs in this group is given in experiment 2 (table III).

A third experiment was started January 29, 1931, which was similar to the preceding two, except that the variation in the dosage of serum was more extreme. Three pigs were given 60 cc each of serum and three were given 20 cc each. All pigs in this experiment showed a definite decrease in the number of leucocytes following the double treatment. The reaction was more pronounced and of longer duration in the low-dose pigs than in the case of the ones receiving a large dose. Detailed record of the counts for the pigs in this group is given in experiment 3 (table III).

The leucopenia, which was observed in the foregoing experiments to be a quite regular phenomenon following simultaneous treatment, in many cases seemed to be followed by a leucocytosis. To make further observations on this point, counts were made

TABLE III—*Leucocyte counts of pigs given simultaneous treatment.**

PIG	WEIGHT (LBS.)	AVERAGE OF 3 COUNTS BEFORE TREATMENT	SERUM (cc)	VIRUS (cc)	COUNTS AFTER TREATMENT (DAYS) 1	2	3	4	5	6	7	8	9
EXPERIMENT 1													
102	75	22,916	35	2	19,296		8,437	9,593	8,593	15,625	24,843	20,312	
103	70	27,629	35	2	22,031		10,000	7,343	13,750	23,593	36,406	43,125	
104	75	26,225	35	2	23,593		16,404	17,500	15,781	34,375	36,406	32,812	
105	75	25,181	35	2	26,406		18,590	17,187	26,406	37,187	42,500	36,562	
EXPERIMENT 2													
154	55	31,979	35	2		25,000	13,000	7,968	15,156	22,187	35,625		
155	60	24,739	35	2		19,218	10,156	10,468	10,625	15,468	28,261		
156	55	26,083	35	2		20,937	9,218	7,656	14,062	20,000	32,500		
157	50	19,322	25	2		13,750	13,906	11,406	10,625	15,781	20,312		
158	50	21,979	25	2		12,812	5,937	6,562	8,750	15,312	20,781		
159	55	24,690	25	2		13,750	9,375	9,062	12,187	12,393	24,375		
EXPERIMENT 3													
164	80	21,779	20	2		12,132	6,150	7,031	8,750	15,781	18,281		22,500
165	75	19,635	20	2		13,281	6,150	5,312	6,718	8,125	13,437		22,500
166	65	19,270	20	2		13,906	7,500	4,218	6,150	10,937	13,281		18,281
167	70	21,510	60	2		22,187	12,968	14,218	12,812	15,625	23,906		
168	75	22,968	60	2		20,468	11,875	13,281	12,656	13,906	26,875		
169	90	21,718	60	2		20,312	10,781	10,312	13,437	15,468	21,093		

*All of these pigs were apparently normal throughout the experiments.

*All of these pigs were apparently normal throughout the experiments.

TABLE IV—*Leucocyte counts of pigs given simultaneous treatment.*

PIG	WEIGHT (LBS.)	COUNT BEFORE TREATMENT	SERUM (cc)	VIRUS (cc)	COUNTS AFTER TREATMENT (DAYS)							
					1	3	4	6	8	10	12	14
109	80	19,531	20	2	18,906	8,437	7,031	7,031	12,812	17,187	16,552	20,000
111	65	14,218	20	2	17,812	11,250	9,375	7,187	13,281	23,437	19,218	20,312
113	70	15,781	20	2	16,718	5,625	8,906	8,125	15,937	15,937	16,250	20,625
117	65	15,156	20	2	10,625	7,968	7,187	8,437	12,812	15,937	16,406	18,281
Averages		16,171			16,015	8,320	8,125	7,695	13,710	18,124	17,109	19,804

on four pigs which were being used in a serum test. By referring to table IV, it will be noted that three of the four pigs in this group showed higher counts at the end of the experiment, on the fourteenth day, than they did on the day it was started. This experiment also furnishes additional evidence that leucopenia follows the simultaneous treatment. The pigs in this test received only 20 cc each of serum, whereas the field dose for pigs of this weight would have been 35 cc. During the reaction period, the white cell counts on all of these pigs reached a point below 8,000 per cmm. for at least one day. Counts were not made daily, but they were continued for a longer period than in the preceding experiments. During this test, the pigs showed no rise in temperature or any other symptoms of disease.

Summary and Conclusions

In summarizing the results of this work, it is noted that in a large number of cholera-infected pigs the average white cell count tends to be lower as the disease progresses. It will be noted also that in many cases, which were undoubtedly hog cholera, leucopenia was not well-marked at all times during the course of the disease. While diseases other than hog cholera have not been extensively studied, a well-marked leucopenia was noted only in cholera-infected pigs. A leucopenia may therefore be regarded as a usual though not constant feature of hog cholera.

In a group of twenty-eight simultaneously treated pigs, there was observed in all cases a distinct, although apparently transitory, leucopenia following this treatment. It appeared also that the severity of the leucopenia was in a measure related to the dose of serum, the leucopenia being more marked in the pigs receiving the lower dosage.

Acknowledgment

I wish to acknowledge my indebtedness to Dr. M. Dorset and Dr. C. N. McBryde for their suggestions in connection with this study.

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A trio of members represented the Gopher State: Drs. C. P. State: Drs. Horst Schreck, El Paso; N. M. Wheeler, Winnsboro; N. F. Williams, Fort Worth.

THE OCCURRENCE OF BRUCELLA AGGLUTININS IN CATTLE IN THE PANAMA CANAL ZONE*

By EDWIN O. JORDAN *and* J. MCBROOM
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A collection of serum from the Mindi Dairy herd in the Panama Canal Zone, in February, 1931, afforded an opportunity for determining the presence of Brucella agglutinins in cattle in this region.† The herd consisted of purebred and grade Holstein and Jersey cattle. The majority of these animals were born on the Isthmus; those brought in from the United States (mostly purebred Holsteins) were from 2 to 6 years old. The herd was under constant and expert veterinary care. The serum was drawn with antiseptic precautions and kept at ice-box temperature except for the few days it was in transit to the laboratory. Phenol (0.5 per cent) was added as a preservative. The usual macroscopic method was used; the mixture of saline suspension and serum was incubated at 37° C. for 2 hours and then kept at ice-box temperature overnight before the readings were made. The period of testing was unavoidably extended over several months, but a number of sera were tested at the beginning of the period and then retested later; the results show that in some cases a slight loss of agglutination strength occurred but that the general tabulations were not affected. Six Brucella strains were used in preliminary tests. One of them, of porcine origin, proved so sensitive that it agglutinated to some degree with all sera tested; another, a human strain, also showed a tendency to agglutinate in relatively high dilutions. As other observers have found, the selection of suitable antigens is a matter of primary importance. Two strains, one from the liver of an aborted pig, obtained from Dr. J. W. Connaway, University of Missouri, and the other from an aborted bovine fetus, obtained from the U. S. Bureau of Animal Industry, Washington, D. C., were finally isolated and gave uniform and consistent results throughout. Rarely one of these strains would be agglutinated at a 1:40 dilution while the other was not, but in almost all instances the readings were identical. Living antigens were used in all tests, partly for the purpose of being able to correlate the results with those of another serological study

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which was in progress in which this procedure seemed desirable. The turbidity of the antigen suspension was kept uniform throughout by comparison with a barium sulfate suspension. Platings were made at frequent intervals to insure smoothness. Comparative tests made with a standard Brucella antigen, obtained from Dr. K. F. Meyer, showed no essential differences in the agglutination titres. The sera of 383 cows were examined, with the results shown in table I.

TABLE I—*Summary of agglutination tests.*

Agglutination Reaction	Animals	
	No.	%
Positive in dilution of 1:100 or higher	52	13.6
Positive in dilution of 1:40, but not at 1:100	43	11.2
Negative in dilution of 1:40	288	75.2
Totals	383	100.0

These results are quite similar to those obtained in other localities, but show a somewhat lower proportion of positive reactions than those recorded, for example, by Norton and Pless,[1] in Michigan (21 per cent positive), or by Hardy *et al.*,[2] in Iowa (26 per cent positive, i. e., 1:80 or higher). The exact comparative results are shown in table II.

TABLE II—*Results obtained in three different localities.*

Locality	Cows Tested	Positive						Negative	
		High Dilution			Low Dilution				
		No.	%	Titre	No.	%	Titre	No.	%
Panama Canal Zone	383	52	13.6	1:100+	43	11.2	1:40	288	75.2
Michigan	647	136	21.0	1:100+	115	17.8	1:25 1:50	396	61.2
Iowa	1300	339	26.0	1:80 +	105	8.0	1:40	856	66.0

If a positive agglutination reaction in a dilution of 1:100 or higher is taken as evidence of infection with Brucella, and absence of reaction in a dilution of 1:40 as evidence of freedom from infection, then the dairy cows in the Canal Zone appear to have been somewhat less generally infected at the time of this test (February, 1931) than those in some other localities.

Enough serum was available for determining the upper titre limit of the sera in 46 of the 52 cases in which agglutination occurred at 1:100 and over. The results are given in table III.

TABLE III—*Upper titre limits of 46 reactors.*

UPPER TITRE LIMIT	REACTORS
1:100	19
1:200	10
1:400	6
1:800	8
1:1600	1
1:3200	2
Total	46

Twenty-two of these 46 sera gave a very definite zoning effect and a few others gave indications of zoning with one or more strains. This was often very marked, as is shown by the examples in table IV. Several zoning sera were treated with the prepared antigen sent us by Dr. Meyer and gave similar results.

TABLE IV—*Zoning effect shown by four sera.*

SERUM	DILUTION					
	1:40	1:100	1:200	1:400	1:800	1:1600
170	−	+	++	tr	.....	.....
133	−	++	+++	+++	tr	−
135	−	tr	+	+++	+	−
144*	−	−	++	++	.....	.....

*In nearly all instances where zoning was observed, the dilution 1:100 showed at least a trace of agglutination. Serum 144 is an exception.

Exact histories with respect to abortion were available for 329 of the 383 cows. Sixty-nine of these had records of from one to three abortions; 260 had no history of abortion. Of the 69 aborting animals, 37.7 per cent gave a serum reacting in a dilution of 1:40 or higher (10.1 per cent, 1:40; 27.5 per cent, 1:100 or higher). Of those with no history of abortion, 21.5 per cent gave a serum reacting at 1:40 or higher (8.8 per cent, 1:40; 12.7 per cent, 1:100 or higher). The occurrence of agglutinins in the serum of cows that have never aborted has been noted by Norton and Pless,[1] Huddleson and Smith,[3] and others. The sera of 14 bulls were tested; all were negative in a 1:40 dilution. The sera of 12 yearling heifers were tested and proved

negative in a 1:40 dilution in 10 cases and positive at 1:40 (and not higher) in 2.

The sera of 28 men, engaged in handling milk and caring for the animals, etc., were tested; all but one were negative in a 1:40 dilution. The single positive reactor (D. G.) was a man about 62 years old who had been a cattleman all his life and was at the time employed in handling the breeding herd and calves. He gave no definite history of undulant fever but had been in the Ancon Hospital, with diagnoses, on separate occasions, of malaria complicated with bronchitis, and amoebic dysentery. No case of undulant fever, traceable to cattle, seems yet to have been recorded in the Zone.

SUMMARY

Brucella abortus infection, as determined by the agglutination test, is present in dairy cattle in the Panama Canal Zone, although apparently to a somewhat less degree than in many parts of the United States. Reactions at 1:100 and higher are more than twice as common in animals that have aborted as in those with no history of abortion. Undulant fever in man, traceable to cattle, has not yet been recorded in the Zone.

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Artifical Sheep Moulting in Russia

A new method of artificial moulting at present in use in Russia is said to have made shearing obsolete, according to a report by the Riga correspondent of the *London Times*, who states that the fleeces of sheep and the fur of rabbits are being gathered four times a year. The Soviet Commissariat of Light Industry describes the process as follows:

> "The sheep are given a small dose of a preparation of heavy mineral salt, which so acts upon the sympathetic nerve-system that in a few days the fleeces are 'loosened' and may be peeled off. No wool is left on the sheep's body, which is described as being smooth and shiny as a man's bald head after the completion of the process. A new fleece begins to grow immediately, and its rate of growth is more rapid than after shearing.
>
> "Professor N. A. Ilyn, who is responsible for this new method, claims that he can make a sheep moult in two stages; first the finest parts of its fleece, then that of coarser texture. Thus even the roughest sheep may be made to yield some fine wool, which is sorted as it is peeled. In the same way, fine rabbit-down is sorted from the coarser fur. The system is also to be applied to the coats of dogs."

STATISTICAL OBSERVATIONS INVOLVING WEIGHT, HEMOGLOBIN AND THE PROPORTION OF WHITE BLOOD CELLS IN PIGS*

By W. A. CRAFT

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and

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This report is based on observations of pigs, which were primarily used for other experimental studies, at the Oklahoma Agricultural Experiment Station during 1931. These pigs include nine litters, some of which were inbred Duroc Jersey and others were crossbred Poland China-Hampshire. The pigs were farrowed in a farrowing-house equipped with brick and cement floors. They had access to a feed mixture, in a creep, consisting of ground yellow corn, wheat middlings, tankage, alfalfa meal and 1.5 per cent mineral mixture which consisted of ground limestone, steamed bone meal, sodium chlorid, ferrous sulfate and copper sulfate. At the end of three weeks they were removed from the farrowing-house and placed on Sudan pastures. The pigs were continued on the same feed until 60 days old, when they were weaned and put on a self-feeder containing the feeds mentioned.

Data here reported are for determinations made at birth and each subsequent 30 days until 120 days old; and in addition, determinations were made at 180 days. Hemoglobin determinations were made by a modification of the Newcomer method, and the proportion of white blood cells was determined by microscopic examination of smears.

Although additional pigs were entered at intervals during the course of these observations, the trend for individuals followed from birth to 180 days is the same as that shown in the tables.

The means for hemoglobin and the proportion of white blood cells are recorded in table I. The hemoglobin is given as the number of grams of hemoglobin per 100 cc of blood; and the white blood cells are shown in percentages.

The statistical constants shown in table II were calculated according to the method given by Wallace and Snedecor.[1]

*Received for publication, May 5, 1932.

While the coefficients are not large enough to be distinctly significant according to accepted tests for significance, with the limited degrees of freedom available, our study of the data causes us to think that the results are of interest. This report is given in the hope that the results may be of value to others who are working on hematological problems.

TABLE I—*The mean for the different observations.*

PIGS		WEIGHT (POUNDS)	MEAN HB.	POLYMORPHONUCLEAR %	LYMPHOCYTES %	EOSINOPHILS %
AGE	NO.					
Birth	32	3.63	9.69	54.90	44.8	0.1
30 days	39	12.20	11.56	34.07	62.3	3.7
60 days	43	26.60	12.75	27.30	63.4	9.2
90 days	44	40.10	13.26	34.40	61.0	4.5
120 days	35	55.40	12.92	41.10	55.05	3.57
180 days	24	137.50	12.58	30.60	64.90	4.37

TABLE II—*Coefficients of correlation for weight and the blood constituents observed.*

PIGS		WEIGHT AND HB.	WEIGHT AND POLYMORPHONUCLEARS	WEIGHT AND LYMPHOCYTES	WEIGHT AND EOSINOPHILS
AGE	NO.				
Birth	32	−0.19	−0.41	+0.04	Not. calc.
30 days	39	−0.19	−0.30	+0.33	−0.22
60 days	43	+0.24	−0.37	+0.33	+0.24
90 days	44	−0.09	−0.33	+0.11	+0.54
120 days	35	+0.44	−0.34	+0.38	−0.17
180 days	24	+0.01	−0.18	+0.21	+0.19

It is of striking interest that weight and hemoglobin show a negative relationship at birth and at 30 days. Between the 30- and 60-day periods, the relationship changed to positive, and for the other periods observed it appears to have a positive trend, although it dropped to negative at the 90-day period. Negative coefficients were found at each period for weight and polymorphonuclear cells; and positive coefficients were obtained for weight and lymphocytes. The relationship for weight and eosinophil cells shows somewhat the same degree of irregularity as weight and hemoglobin. The eosinophil cells showed a marked tendency to decrease in number after the pigs were treated for worms. This is evident in table I. The pigs were treated immediately after the 60-day bleeding.

If weight is negatively correlated with the number of polymorphonuclear cells and positively correlated with the number

of lymphocytes, a negative relationship would be expected between these two classes of cells. This was found to be the case and the coefficients are significantly high. Further study is needed to determine the value of the relationships suggested by these coefficients.

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[1]Wallace and Snedecor: Correlation and Machine Calculation. Off. pub., Iowa State Coll., rev. ed. (1931).

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BREEDING HISTORY AND GROSS CHANGES FOUND ON AUTOPSY IN THE GENITAL ORGANS OF DAIRY CATTLE*

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R. R. GRAVES
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When an animal is no longer of value for experimental work at the United States Dairy Experiment Station at Beltsville, Md., it is killed and the body and organs handled in accordance with the experimental routine of a project on the relation of conformation and anatomy to producing capacity. Observations on the macroscopic and pathological changes in the genital organs and breeding history of 83 females killed at the Station between September 9, 1926, and December 31, 1931, are given here.

Of these 83 animals, 16 were killed while pregnant or shortly after the termination of pregnancy and may be considered as having been normal at the time. Of the remaining 67 animals, 5 had never calved and 17 aborted their last pregnancies, leaving 45 that had terminated their last pregnancies normally. Eight of these 67 animals were considered to be fertile and 59 permanently sterile at the time of slaughter.

In the 5 females that never calved, 144 estrual cycles, varying in length from 4 to 257 days, were observed. These animals were bred from 11 to 23 times each without conceptions. Antemortem examinations revealed the presence of an infantile uterus in one animal, but no abnormalities were found in the four others.

When posted, the genital organs of two (40 per cent of these 5 animals) appeared normal; one showed an infantile uterus; one lacked an opening through the cervix, and one showed adhe-

*Received for publication, July 23, 1932.

sions and fibrous growth around the anterior part of the uterus and ovaries, evidently a result of inflammation from enteritis and pneumonia during calfhood.

Between the termination of the last pregnancy and the killing, the 17 animals that aborted showed 275 estrual cycles, varying in length from 2 to 238 days. These animals were bred from 3 to 23 times each without conceptions. Three of the abortions were followed by retained placenta. Antemortem examinations revealed a lack of muscular tone in 7 of these cows; adhesions in one; large ovaries in one; an abscess on the uterus in one; tumors interfered with examination in one, and no abnormalities were found in the 6 others.

When these 17 animals were killed, the genital organs of 3 (17.64 per cent) appeared normal; 9 (52.94 per cent) showed inflammatory changes caused by spread of inflammations from elsewhere in the body; 2 (11.76 per cent) showed inflammatory changes originating within the genital organs; and 3 (17.64 per cent) showed cysts in some part of the genital organs.

In the 45 females that were killed some time after having given birth to apparently normal calves, 565 estrual cycles, varying in length from 2 to 338 days, were observed. Only one animal had regular estrual cycles of 20 or 21 days. Retained placenta was found in 7 of these animals following birth of last normal calf. Of these 45 animals, 8 had not been bred, while the other 37 had been bred from one to 24 times each without conceptions. Antemortem examinations revealed a lack of muscular tone in 10 of these cows; adhesions in one; large ovaries in 7, also in 3 following metritis; ovarian cysts in 5; cervicitis in 2; enlarged uterus in 2; reabsorption of fetus in 2; tumors interfered with examination in 2, and no abnormalities were found in the 9 others.

Autopsy revealed that 6 (13.33 per cent) of these animals had apparently normal genital organs; 25 (55.55 per cent) showed inflammatory changes caused by spread of inflammations from elsewhere in the body; 7 (15.55 per cent) showed inflammatory changes originating in the genital organs; and 7 (15.55 per cent) showed cysts in some part of the genital organs.

Of the 8 animals considered as fertile, 7 had apparently normal genital organs when posted and one showed inflammatory changes that evidently were started by a foreign body. Four of the 59 animals considered as sterile showed apparently normal genital organs on autopsy; 2 showed anatomical deficiencies; 34 showed inflammatory changes originating elsewhere in the body

(the inflammatory changes in 13 of these could be traced directly to foreign bodies, 3 to fat decomposition tumors, 2 to calfhood pneumonia, and 16 to inflammations of unknown origin); 9 showed endometritis, and 10 showed cysts in some part of the genital organs. Of the 10 cows that had retained placenta, 6 showed inflammation and 4 revealed cysts.

SUMMARY

A study of the gross changes of the genital organs in the cows reported here indicates that sterility in the majority of cases was due to inflammations involving the genital organs. The genital organs of only four sterile animals appeared normal.

POSTERIOR PARALYSIS DUE TO A FRACTURED VERTEBRA*

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Michigan Agricultural Experiment Station
East Lansing, Mich.

Subject: A Holstein heifer, aged 16 months.

History: For several months this animal had been on an experimental ration deficient in vitamin D. The day previous to autopsy the heifer was mounted by a young bull in the exercising-yard. Soon afterward the heifer was found in a recumbent position unable to rise. For several hours previous to autopsy, a comatose condition existed.

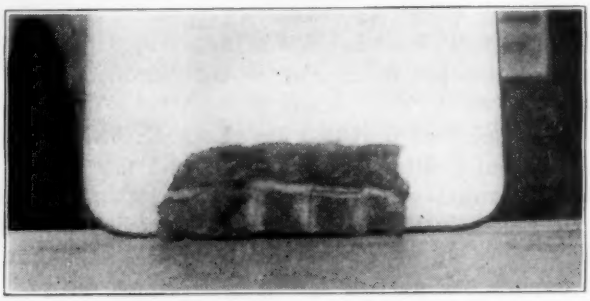

FIG. 1. Fracture of fifth lumbar vertebra resulting in severance of the spinal cord.

Diagnosis: Posterior paralysis probably due to an injured vertebra.

Autopsy: Animal in fair physical condition. Skin and subcu-

*Received for publication, August 1, 1932.

taneous tissues negative. Head and neck negative. Lymph-nodes negative. Chest and thoracic viscera negative. Abdomen and its viscera negative except the kidneys. Each kidney showed a few subcapsular petechiae. Spinal column: a saggital section revealed complete fracture of the fifth lumbar vertebra. The prolapsed portion caused sufficient pressure to sever the spinal cord, as is shown in the accompanying photograph (fig. 1).

The writers have observed similar fractures in animals fed on rations deficient in vitamin D. In some instances the fractures apparently occurred spontaneously.

THE TIME ELEMENT IN REMOVAL OF RETAINED FETAL MEMBRANES*

By C. F. CLARK, *East Lansing, Mich.*
Michigan Agricultural Experiment Station

The writer believes that not enough emphasis has been laid on the time element in discussions of removal of retained fetal membranes. The results in the limited number of cases reported seem to demonstrate that early manual removal is not always wise. Removal of the retained fetal membranes in 48 hours or less after parturition resulted unfavorably in 18 per cent of the cases listed. Removal in 72 to 96 hours after parturition resulted unfavorably in 4 per cent of the cases listed.

The procedure followed in handling the cases reported was as follows: If the fetal membranes were not passed within 24 hours after parturition, an examination was made. If the membrane was retained by less than four placental attachments, it was removed. If the placentitis was extensive, and a majority

TABLE I—*Data on 47 cases of retained placenta.*

WHEN REMOVED	RECOVERY COMPLETE	RECOVERY INCOMPLETE OR FAILED
24 hours after parturition	9	
48 hours after parturition	5	4†
72 hours after parturition	15	
96 to 120 hours after parturition	12	2†
Totals	41	6

*Received for publication, August 1, 1932.
†One cow in each of these groups aborted and exhibited symptoms of septicemia from the time of abortion; both died.

or all of the fetal placental areas tightly adherent, no effort at removal was made, but antiseptics were introduced. The membranes were removed when the fetal placental villi had softened enough to slip readily from the crypts of the maternal placental area. From a study of table I it would appear that 72 to 96 hours was the optimum time for removal.

An explanation should be made concerning the first line in table I lest it give a false impression. It states that all of the 9 cows from which the membranes were removed in 24 hours made a complete recovery. It should be remembered that in these cases the membrane was retained by only a few placental attachments, and in some cases only one. In none of the nine cases was the condition extensive, as was true in all of the remaining 38 cases reported. Complete recovery means that the cow later conceived. In the last column, headed "Incomplete recovery or failure," two cows failed to conceive, two died, and two were slaughtered.

Doctor Dimock Called to England Again

Dr. W. W. Dimock, of the University of Kentucky, went to England again in July, at the invitation of the British Bloodstock Agency, Ltd., of London. The object of the trip was to repeat the work inaugurated last year for the handling of breeding diseases at Thoroughbred studs. Visits to some of the largest breeding establishments in England, Ireland and France were included in Dr. Dimock's itinerary.

Meat Inspection in Scotland

New meat inspection regulations in Scotland include the following ruling:

> "No person is qualified to act as a meat inspector—unless he is either the Medical Officer of Health of the area, a veterinary surgeon, or a person who has received special training in the work of meat inspection, and prior to June 1, 1923, has had not less than 7 years' practical experience in that work, and has obtained a certificate from the Department."

Rabies Quarantines

All dogs in Marshall County, Tenn., were placed in quarantine for a period of ninety days, on August 17, on account of the existence of rabies in the county. A 90-day quarantine was placed on all unvaccinated dogs in Wellston, Ohio, August 24.

ENDOCARDIAL CALCIFICATION IN DOMESTIC ANIMALS. W. S. Tscherniak and N. Romanov. Abst., Arch. Path., xiii (1932), 4, p. 664.

In 9 cases of calcification of the parietal endocardium, in 238 horses and 3 dogs, the process attacked chiefly the endocardium of the left ventrical and was associated with calcification of the walls of the blood-vessels and with a degenerative process of the endocardium in general.

THE EFFECT OF TOXIC SUBSTANCES OF THE TUBERCLE BACILLI ON THE LIVER. A. M. Lewin. Abst., Arch. Path., xiii (1932), 4, p. 668.

Lipoid and albuminous endotoxic substances of the tubercle bacillus were injected into rabbits, and the changes in the liver and spleen studied at intervals varying from 10 days to 12 months. One hundred and seventy histologically specific tuberculous changes were found. In the liver there occurred a connective tissue hyperplasia that led to a cirrhosis much like the common interlobular cirrhosis of man. In the spleen the connective tissue was increased and the sinus endothelium was hyperplastic. The findings support the view that tuberculous intoxication may play an important rôle in the pathogenesis of interlobular cirrhosis.

SPREAD OF RABIES VIRUS WITHIN THE BODY. F. Schweinberg and F. Windholz. Abst., Arch. Path., xiii (1932), 4, p. 670.

Experimental and histologic evidence has established the transfer of rabies from the point of introduction to the central nervous system by way of the nerve-paths. Whether the virus may not also be transported by the blood-stream, for which some evidence has been offered, has not been determined in equally definite manner. The authors used the method of parabiosis. That a communication between the circulations of two united

rats has occurred by the sixth day after union could be proved by the subcutaneous injection of an aqueous solution of methylene blue into one of the pair. The dye appeared in the urine of the other as soon as in that of the animal receiving the injection. Circulatory union having been proved in this manner, fixed rabies virus was injected intramuscularly into the thigh of one of the parabiotic rats. In each of the eighteen experiments rabies virus could be demonstrated only in the animal receiving the injection.

THE MAINTENANCE OF A NORMAL PLASMA PROTEIN CONCENTRATION IN SPITE OF PROTEIN LOSS BY BLEEDING. C. W. Barnett, R. B. Jones, and R. B. Cohn. Jour. Exp. Med., lv (1932), 5, p. 683.

Experiments on five dogs are described, consisting in the daily removal of blood-plasma in amounts from 25 to 100 cc, the red cells being returned to the circulation in Locke's solution. In no case was there a significant drop in plasma protein concentration. A gravimetric method for the determination of total plasma protein is described. A case is reported of cirrhosis of the liver in which over ten grams of protein daily was lost in the ascitic fluid during a period of seven months without any lowering of plasma protein concentration. The constancy of the plasma protein level and the adequacy of the mechanism of regeneration is pointed out.

REACTIONS OF RABBITS TO INTRAVENOUS INJECTIONS OF PNEUMOCOCCI AND THEIR PRODUCTS. VII. THE RELATION OF HYPERSENSITIVENESS TO LESIONS IN THE LUNGS OF RABBITS INJECTED WITH PNEUMOCOCCI. L. A. Julianelle and C. P. Rhoads. Jour. Exp. Med., lv (1932), 5, p. 797.

The intratracheal injection of egg albumin or pneumococci protein induces an inflammatory reaction in the lungs of rabbits previously inoculated with the respective antigen. A similar reaction occurs following intratracheal injection of pneumococci protein into the lungs of rabbits previously inoculated with heat-killed suspensions of bacteria. This reaction appears to be related to the presence of circulating antibody and to have the nature of the Arthus reaction. A study of the reaction of the lungs of rabbits to infection caused by intravenous injections of pneumococci reveals that (a) reactions occur regularly in the lungs; and (b) in the lungs in which reactions do occur, the

histological changes are not different in normal rabbits and in rabbits made resistant by previous intravenous and intracutaneous injections of pneumococci. Intratracheal injection of pneumococcus protein followed by intravenous injection of virulent pneumococci on the next day does not alter the course and character of the infection in resistant rabbits.

PULLORUM DISEASE IN POULTS. Ralph P. Tittsler. Poultry Sci., xi (1932), 2, p. 78.

The author describes an outbreak of pullorum disease in a group of young poults, the first symptoms appearing when the birds were but three days old. *Salmonella pullorum* was isolated from the heart and liver of the two birds autopsied. The symptoms resembled those of the same disease in the baby chick. The surroundings were cleaned thoroughly twice a day using a chlorin-liberating disinfectant and the birds were fed liberally upon liquid skim milk, the disease disappearing within two to three days. It is suggested that the infection originated either in the breeding stock or in the incubator.

STUDIES OF DISEASES OF THE LYMPHOID AND MYELOID TISSUES. IV. SKIN REACTIONS TO HUMAN AND AVIAN TUBERCULIN. Frederic Parker, Jr., Henry Jackson, Jr., Greene Fitzhugh and Tom D. Spies. Jour. Immunol., xxii (1932), 4, p. 277.

Tuberculin tests using both human and avian tuberculin were carried out on patients with Hodgkin's disease, malignant lymphoma, leukemia, pernicious anemia, and malignancy. Similar tests were done on tuberculous and normal individuals. Fewer positive reactions were obtained in the patients with the diseases of the lymphoid and myeloid tissues and with malignant disease as compared with the normals and the tuberculous patients. More positive reactions to avian tuberculin than to human were found except in the normal group.

CHEMICAL STUDIES OF THE BLOOD IN HIGH INTESTINAL OBSTRUCTION. I. PHOSPHORUS AND INTRACELLULAR CHANGES. George Martin Guest and William DeWitt Andrus. Jour. Clin. Invest., xi (1932), 3, p. 455.

Following experimental pyloric and mid-duodenal obstruction in dogs, marked changes in the phosphorus in the blood has been observed. The phosphorus was partitioned as in the following fractions in the whole blood, plasma and cells: inorganic-acid-

soluble; organic-acid-soluble or "ester P"; acid-insoluble; alcohol-ether-soluble or lipin P; total phosphorus. The most important changes were marked increases in the fraction designated as "ester P" which has an average normal value of 50 mgm. per 100 cc in the cells and only 0.3 mgm. per 100 cc in the plasma. The increases of the "ester P" were much greater than the changes in any of the other phosphorus fractions of the cells or plasma. Changes in chlorid and carbon dioxid content of both plasma and cells were compared with concomitant changes in the phosphorus. In all of the experiments there was a close correlation between the progressive losses of chlorid from the blood cells and the increases of organic-acid-soluble phosphorus. The parenteral administration of sodium chlorid solution to obstructed dogs prevented the increases of organic-acid-soluble phosphorus in the blood cells to about the same degree that it prevented the losses of chlorid from the blood-cells.

CHEMICAL STUDIES OF THE BLOOD IN HIGH INTESTINAL OBSTRUCTION. II. THE RELATION BETWEEN TOXEMIA AND CHEMICAL CHANGES. William DeWitt Andrus, George M. Guest, Richard F. Gates and Alta Ashley. Jour. Clin. Invest., xi (1932), 3, p. 475.

In dogs with pyloric obstruction, repeated subcutaneous injections of small doses of histamine have the effect of hastening the development of those chemical changes in the blood which have been claimed by many to be the most important ultimate cause of death. Histamine was employed as a known substance which is at least closely related to the toxic substances which appear in the contents of a segment of obstructed or strangulated bowel. Histamine, injected subcutaneously as in these experiments, stimulates the flow of gastric juice and, in the presence of vomiting, such increased secretion results in more rapid losses of electrolytes and water than occur in animals with simple obstruction; in consequence of this there is a more rapid development of the whole cycle of the symptoms and chemical changes which ordinarily accompany intestinal obstruction. From these experiments it is suggested that dogs, with experimental obstruction plus closed loops, die sooner than do those with simple obstruction, not because of any specific general intoxication of the body tissues, but because the slow absorption of histamine-like substances from the bowel results in an acceleration of the progress of all those chemical changes which occur secondarily to the losses of gastric secretions.

STUDIES IN THE PHARMACOLOGY OF METAPHEN AND ACRIFLAVINE. P. Jeanette Crittenden, Jour. Phar. & Exp. Therap., xliv (1932), 4, p. 423.

Metaphen intravenously, except in enormous doses, does not have any effect upon the blood-pressure, heart-rate and respiration of either barbitalized or unanesthetized dogs. In general, intravenous injection of therapeutic doses (0.15 gm. per kilo) caused a marked leucocytosis accompanied by a proportional increase in the different forms of leucocytes. In one out of ten dogs a leucopenia resulted. It has no effect in similar doses on the fragility of erythrocytes. The minimum lethal dose of metaphen in dogs is 3.5 mgm. per kilo body weight, which, on the basis of its mercury content, is equivalent to the m. l. d. of bichlorid of mercury. Certain preparations of acriflavine are definitely toxic, causing marked changes in the heart-rate, circulation and respiration, and nausea and vomiting in intravenous doses of from 2 to 5 mgm. per kilo body weight. A neutral preparation of acriflavine can be prepared, which, in doses of from 1 to 2 mgm. per kilo, causes either no or only slight changes in heart-rate, respiration and blood-pressure. The least toxic preparation of acriflavine in small doses stimulates and in larger doses depresses the peripheral vagal mechanism of the heart. The chief point of action of acriflavine on the blood-pressure is peripheral.

ANATOMICAL CHANGES IN THE LIVERS OF DOGS FOLLOWING MECHANICAL CONSTRICTION OF THE HEPATIC VEINS. J. P. Simonds and J. W. Callaway. Amer. Jour. Path., viii (1932), 2, p. 159.

The livers of dogs examined 24, 48 and 72 hours and 7 days after mechanical obstruction of the hepatic veins for periods of 7 to 30 minutes showed the following changes: a mean increase of 25 per cent in the liverweight-bodyweight ratio, due to edema and to swelling of the hepatic cells; swelling, granulation, vacuolization and extensive necrosis of the hepatic cells in the central half or two-thirds of the liver lobules; marked dilatation of the perivascular lymphatics surrounding the sublobular veins; the presence of hyalin thrombi in many central and sublobular veins; intrasinusoidal cell masses of two types: (1) small compact occluding masses, probably originating in "conglutination thrombi" of red cells, and (2) larger, more diffuse and branching cell masses; hemosiderosis of Kupffer cells.

A STUDY OF THE INTESTINAL FLORA OF CHICKS AFFECTED WITH PULLORUM DISEASE. M. W. Emmel. Jour. Inf. Dis., 1 (1932), 3, p. 213.

In a systematic study of the intestinal contents of thirty pullorum-positive chicks, colon organisms constituted an average of 32.35, 38.02 and 63.35 per cent of the bacterial flora of the duodenum, middle portion and cloaca, respectively. In the same study *Salmonella pullorum* constituted an average of 47.26, 47.61 and 19.63 per cent of the bacterial flora of the duodenum, middle portion and cloaca, respectively. The inoculation of 24-hour broth cultures of *S. pullorum* with colon organisms resulted in a reduction in the number of *S. pullorum* per cubic centimeter from 189,000,000 to 32,000,000 after incubation for 24 hours. The hypothesis is advanced that a colon flora is beneficial in the intestinal tract of the chick. In thirteen of fifteen chicks that recovered from pullorum disease, *S. pullorum* persisted in the feces for one week, in eight chicks for two weeks, in three chicks for three weeks, and in one chick for five weeks, after the climax of the outbreak.

B. A. I. Veterinarians Retired

The so-called economy bill, passed at the recent session of Congress and signed by the President on June 30, provided that no person in civilian service in any branch of the U. S. Government, who had reached the retirement age prescribed for automatic separation from the service, could be continued in the service after June 30 except by executive order of the President.

The Department of Agriculture was affected to the extent of 157 employes, of whom 96 were in the Bureau of Animal Industry. Most of this number, including 26 veterinarians, were connected with the meat inspection service. They are:

*Lineus J. Allen
Edwin W. Barthold
George W. Bromell
*Adolph M. Caspar
Wm. Ross Cooper
*Miles L. Davenport
*Harry H. Dell
Oro W. Everly
Abraham J. Farley
*Jonathan E. Gibson
Bartlett E. Harper
*Albert N. Hughes
Moses Isaac
Matthias S. Lantz
Jens Madsen
Louis Metsker
William Mink
*Charles F. Palmer
*Joseph W. Parker
*Thomas B. Pote
*Robert A. Ramsey
*Zachary Veldhuis
*C. Otto Wagoner
*Alex C. Walls
William C. Wooton
John B. Wright

*Member of A. V. M. A.

Regular Army

The following-named officers of the Veterinary Corps are relieved from further assignment and duty at the stations specified after their names, effective in time to comply with this order. They will proceed to Washington, D. C., and report to the commanding officer, Army Medical Center, on or about August 25, 1932, for duty, for the purpose of pursuing a course of instruction at the Army Veterinary School:

Major Seth C. Dildine, Fort Des Moines, Iowa.

Major Charles S. Williams, Fort Reno, Okla., and for additional duty at the purchasing and breeding headquarters, Fort Reno.

2nd Lt. Wesley W. Bertz, Fort Riley, Kan.

The promotion of the following-named officers is announced:

Lt. Col. Robert C. Musser to the grade of colonel, ranking from July 24, 1932.

Captains Claude F. Cox and Harry L. Watson to the grade of major, ranking from June 28 and July 18, 1932, respectively.

2nd Lt. Harvie R. Ellis to the grade of 1st lieutenant, ranking from June 24, 1932.

Par. 4, S. O., 99 W. D., 1932, relieving Major Raymond A. Kelser, Veterinary Corps, from assignment and duty at the Army Medical School, Army Medical Center, Washington, D. C., from additional duty at Ft. Myer, Va., and directing him to proceed to Boston, Mass., for duty, is revoked.

Major Raymond T. Seymour is relieved from further assignment and duty at Fort Snelling, Minn., effective on or about August 25, 1932, and will then proceed to Lexington, Ky., and report to the officer in charge remount purchasing and breeding headquarters for duty.

Veterinary Reserve Corps

New Acceptances

Allen, Raymond R..2nd Lt..Box 118, R. F. D., Leominster, Mass.
Berger, Samuel J..2nd Lt..961 Faile St., Bronx, New York, N. Y.
Betzold, Curtis Wm.2nd Lt..1409 Seventh St., Sanger, Calif.
Blostein, Morris E..2nd Lt..411 Cascadilla St., Ithaca, N. Y.
Buckley, Bernard T.2nd Lt..Maurice, Iowa
Ditman, Noran Lyle.2nd Lt..Stanton, Neb.
Dugan, Lester V...2nd Lt..R. F. D. 2, Minburn, Iowa
Eldred, Arthur C..2nd Lt..Bombay, N. Y.
Gaydosh, Louis W..2nd Lt..P. O. Box 282, Fords, N. Y.
Geisler, Richard E..2nd Lt..3555 Woolworth Ave., Omaha, Neb.
Gifford, Ralph W..2nd Lt..R. F. D., E. Bethel, Vt.
Gingras, George E..2nd Lt..252 W. Main St., Gouverneur, N. Y.
Granholm, Paul R..2nd Lt..7 S. Jefferson Ave., Mason City, Iowa
Lange, Chester J...2nd Lt..11 Hinchman Ave., Dover, N. J.
Merchant, W. R....2nd Lt..Garden Grove, Calif.
Peterson, Alfred....2nd Lt..Box 604, Kenmare, N. Dak.
Pieper, Niels W....2nd Lt..Francis Ave., Newington Jct., Conn.

Presler, Donald J...2nd Lt..Prattsburg, N. Y.
Ranney, Albert F...2nd Lt..Putney, Vt.
Sadler, Edw. G., Jr.2nd Lt..Pine Plains, Dutchess Co., N. Y.
Schladweiler, A. J..2nd Lt..Madison, Minn.
Schoneman, Ben E.2nd Lt..George, Iowa
Stalnaker, H. B....2nd Lt..Edgewood, Iowa
Sternfels, Mark.....2nd Lt..560 W. 180th St., New York, N. Y.
Stevenson, John C.2nd Lt..17 Butler St., Westbury, N. Y.
Tabbut, Herbert M.2nd Lt..R. F. D. 2, Pelican Rapids, Minn.
Torrey, J. Philip...2nd Lt..Animal Pathology Lab., Urbana, Ill.
Garrett, Thos. W...2nd Lt..208 W. 8th Ave., Columbus, Ohio
Holmes, John M....2nd Lt..403 16th Ave., Columbus, Ohio
Karr, James R.....2nd Lt..R. F. D. 3, Coshocton, Ohio
Knapp, John H.....2nd Lt..R. F. D. 4, Lebanon, Ohio
Neuenschwander,
L. F.....2nd Lt..R. F. D. 3, LaRue, Ohio
Rooks, Elmer Lyle..2nd Lt..R. F. D. 1, Greenfield, Ohio
Tanner, Warren L..2nd Lt..Napoleon, Ohio
Treat, Lester A....2nd Lt..R. F. D. 1, Blandford, Mass.
Hoskins, Robt. J....2nd Lt..R. F. D. 1, Wilmington, Ohio
Batchelder, Ray M..2nd Lt..2125 Peasley St., Columbus, Ohio
Bates, Morgan W...2nd Lt..R. R. 4, Washington Court House, Ohio
Bechtol, Lauren L...2nd Lt..Okolona, Ohio
Collier, James R...2nd Lt..Wilkinson, Ind.
Durigg, John R....2nd Lt..Armstrong's Mills, Ohio
Elwood, Guilford S.2nd Lt..2442 Olentangy Road, Columbus, Ohio
Nixon, Jay Earl....1st Lt..Oxford, Kan.
Thom, Myron A....2nd Lt..816 S. San Pedro St., Los Angeles, Calif.
Flory, Oliver E.....2nd Lt..809 Holland St., Great Bend, Kan.

BUREAU TRANSFERS

DR. IRVIN OWENS (K. C. V. C. '03) from South Saint Paul, Minn., to Ogden, Utah, on stock yards inspection.

DR. W. C. DENDINGER (St. Jos. '16) from Raleigh, N. C., to Atlanta, Ga., in charge of tuberculosis eradication.

DR. J. A. THOMPSON (K. C. V. C. '07) from Grand Forks, N. Dak., to West Fargo, N. Dak., on meat inspection.

DR. OTTO HORNLEIN (McK. '10) from Indianapolis, Ind., to South Saint Paul, Minn., on meat inspection.

DR. C. L. ELLIOTT (Iowa '02) from Newark, N. J., to Indianapolis. Ind., in charge of meat inspection.

DR. A. L. HIRLEMAN (Cin. '03) from Atlanta, Ga., to Raleigh, N. C., in charge of tuberculosis eradication.

DR. G. E. TOTTEN (Chi. '98) from South Saint Paul, Minn., to Chicago, Ill., in charge of meat inspection.

DR. H. BUSMAN (Ont. '95) from Chicago, Ill., to Indianapolis, Ind., in charge of tuberculosis eradication.

DR. W. M. HAAG (McK. '03) from New Orleans, La., to Saint Louis, Mo., on meat inspection.

DR. GEORGE R. HARTMANN (K. C. V. C. '11) from Moultrie, Ga., to Chattanooga, Tenn., in charge of meat inspection.

DR. B. N. LAUDERDALE (A. P. I. '17) from Orlando to Arcadia, Fla., on tick eradication.

DR. J. W. HOVORKA (McK. '19) from Chicago, Ill., to Peoria, Ill., in charge of meat inspection.

NEW YORK STATE VETERINARY MEDICAL SOCIETY

The regular annual meeting of the New York State Veterinary Medical Society was held at James Law Hall, Ithaca, June 29-30, 1932. Dr. A. R. Mann, acting president of Cornell University, welcomed the Society, and Dr. R. S. MacKellar responded. President J. L. Wilder read the annual address.

Papers were then delivered by Dr. R. H. Spaulding on "Nephritis in Dogs," and Dr. F. F. Fehr on "Some Remarks by a Small-Animal Practitioner." A brief discussion followed. It was planned to have the Society hear the radio address scheduled to be given over WGY, by C. B. Heisler, of the Department of Education, but, owing to the Democratic Convention at Chicago being in session, this was impossible. The paper was read later.

At the afternoon session, addresses on "Mastitis" and "Bang's Disease" were given by Drs. G. W. Derrick and R. R. Birch, respectively. These were followed by brief addresses on "The Purpose of the Farm Bureau" and "Program of the Emergency Milk Committee of the New York Milk Shed," after which demonstrations and clinics were held, at the various buildings, on mastitis, the taking of blood samples, surgical cases, small animals, etc.

At the annual dinner that evening, attended by about 175, including 50 ladies, at which Dean-elect Hagan acted as toastmaster, the Society was favored with an address by Charles H. Baldwin, Commissioner-elect of the Department of Agriculture and Markets. Prof. A. A. Allen followed with a very interesting motion-picture, showing the partridge (native grouse) in the act of drumming and other characteristic postures in his native woods. Wayne Dinsmore, secretary of the Horse Association of America, gave an inspiring address on "The Horse Industry," which was given close attention.

The following morning Dr. Maurice C. Hall gave one of his

usual fine talks on "Control of Parasites," which was discussed by many members. G. Emerson Markham, of studio WGY, then spoke on "Radio Publicity for the Profession" and urged a continuation of the Society's weekly program. Dr. R. E. Lubbehusen, of the Pennsylvania Bureau of Animal Industry, then discussed "Important Poultry Diseases" and pointed out their importance in agriculture. Dr. Paul B. Brooks, Deputy Commissioner of Health, referred to the wide field of activity open to the profession in sanitary work, and Dr. F. W. Andrews, chairman of the Executive Board, discussed "Accredited Herd Testing and the Practitioner," and referred to the importance of this work being carefully and conscientiously carried out.

At the business session some very important matters were discussed, including the future broadcasting policy of the Society and a complete revision of the constitution, which will be submitted for final action next year. The new constitution provides for an increase in the dues to $5.00 annually; for an executive budget, and the plan of having an executive board and an executive secretary will therefore be continued. It was voted to contribute $500.00 to the International Veterinary Congress fund and to delegate authority to the Executive Board to consider affiliation with the A. V. M. A. Resolutions were offered and passed with respect to a number of deceased members and the late Dr. Bernhard Bang. A resolution was unanimously passed disapproving of the recognition or employment of unlicensed veterinarians in state, county or municipal work.

Twenty-two applicants were voted into membership. Dr. Wm. Henry Kelly, of Albany, was elected president of the Society for the coming year, and it was voted that the next annual meeting be held in that city. The attendance was in excess of 200, including a considerable number of ladies.

J. G. WILLS, *Secretary.*

NORTH DAKOTA VETERINARY ASSOCIATION

The thirtieth annual meeting of the North Dakota Veterinary Association was held at Bismarck, July 11-12, 1932. The occasion was of special interest, for it was held primarily in honor of Dr. W. F. Crewe, who has served for 25 years as State Veterinarian and as executive officer of the Live Stock Sanitary Board since the passage of the law which created the Board. This law was enacted at the legislative session in 1907 and became effective in April of that year. Its usefulness as the legal basis of

the state control work in dealing with transmissible animal diseases has more than fulfilled the expectations of its sponsors.

The meeting likewise served to celebrate another accomplishment in animal disease control work. The U. S. Bureau of Animal Industry had announced some days previously that North Dakota had been added to the modified accredited tuberculosis-free area. This is the eighth state to complete its program of coöperative work between the Bureau and the state authorities. (See JOURNAL, August, 1932, p. 147.)

The session on the first day was occupied with two papers. "The History of Live Stock Sanitary Control Work in North Dakota" was presented by Dr. W. F. Crewe. This was an interesting review of the development of veterinary control work, with its origin in territorial days. Dr. L. Van Es, of the University of Nebraska, then gave in his usual thorough manner a discussion entitled "Remarks on Certain Mineral Deficiencies."

A banquet session in honor of Dr. Crewe was held in the Patterson Hotel on the evening of July 11. There was an attendance of about 45 and it was an event of outstanding significance. An interesting program of short talks appropriate for the occasion was presented. A number of congratulatory messages and letters, from friends and associates of Dr. Crewe who are interested in live stock sanitary control work, had been received. They were read by title and then presented to Dr. Crewe.

The guests and speakers included Judge John Burke, of the State Supreme Court, who was governor at the time of the passage of the Live Stock Sanitary Law in 1907; P. E. Byrne, who was secretary to Governor Burke at that time; W. L. Richards, president of the Live Stock Sanitary Board, who has been a member of the Board since it was established; Dr. J. W. Robinson, who served as toastmaster, likewise a member of the original Board and at present a member, although his service has not been continuous; Dr. C. H. Hofstrand, junior member of the Board; Dr. E. J. Walsh, a member of the original Board; Dr. E. V. Lagerberg, president of the Association; Dr. J. B. Hollenbeck, U. S. Bureau of Animal Industry, Columbus, Ohio; Dr. R. E. Robinson, State Veterinarian of South Dakota; Dr. H. H. Cohenour, B. A. I. Inspector-in-Charge for North Dakota, and Dr. C. H. Hays, B. A. I. Inspector-in-Charge for South Dakota. One of the features of the evening was an address appropriate for the occasion by Dr. L. Van Es. Dr. Crewe responded to the presentation of a gift from the Association, North Dakota veterinarians and friends.

Dr. W. J. Butler, State Veterinarian of Montana, was unable to arrive in time for the banquet, but brought his greetings to the Association the next day.

A delightful program for the entertainment of the ladies had been provided by the local committee.

Three papers and discussions occupied most of the morning of the second day: "Comparative Medicine. Veterinary Service *versus* Public Health Service," Dr. Robert W. Allen, State Public Health Department; "Swine Disease Control," Dr. J. B. Hollenbeck, U. S. Bureau of Animal Industry, Columbus, Ohio; and "The Fulfillment of Our Bovine Tuberculosis Program," discussed by Dr. Crewe and Dr. Cohenour. Dr. Hollenbeck, as the official representative of the U. S. Bureau of Animal Industry at this event, then presented a brief paper celebrating the accreditation of North Dakota as a tuberculosis-free state.

The officers for the ensuing year are: President, Dr. W. D. Odou, Hettinger; vice-president, Dr. R. R. Cusack, Carrington; secretary-treasurer, Dr. Lee M. Roderick, State College Station, Fargo.

L. M. RODERICK, *Secretary.*

WESTERN NEW YORK VETERINARY MEDICAL ASSOCIATION

The nineteenth semi-annual meeting of the Western New York Veterinary Medical Association was held at Chestnut Ridge Park, near Orchard Park, N. Y., July 14, 1932.

A basket lunch was enjoyed by members and their families at noon. The meeting was called to order by President F. L. Stein at 2:30, forty members responding to the roll-call. Routine business was transacted, after which the papers were taken up.

Dr. John Sturrock, of Attica, N. Y., offered a very interesting paper on "Teat Operations," followed by Dr. C. L. Kern, of Syracuse, N. Y., head of the Dairymen's League, on "Milk Pasteurization and Milk Control." Dr. C. M. Carpenter, of Rochester, N. Y., gave a short talk on research work at the Strong Memorial Hospital, in Rochester.

Dinner was served at 6:30, at the Casino in the park, to seventy-five persons, including members, their wives and guests.

After the repast Dr. R. Gwatkin, of Toronto, Ont., spoke on the work being done on Bang's disease and other branches of investigation at the Ontario Research Foundation, located in Toronto.

The final talk was given by Dr. F. W. Graves, of Albany, N. Y., on "Milk Control."

Beautiful weather, a fine crowd, good speakers and good things to eat made this one of the most enjoyable of the meetings of the Association.

F. F. FEHR, *Secretary.*

VERMONT VETERINARY MEDICAL ASSOCIATION

The twenty-fourth annual meeting of the Vermont Veterinary Medical Association was held at the K. of C. Clubhouse, Saint Albans Bay, Vt., July 14-15, 1932, with about forty veterinarians in attendance, including members and visitors.

The meeting was called to order by President William A. Hamilton, and the following papers were presented: "Mastitis Field Work," by Dr. C. W. Gates, Plattsburg, N. Y.; "Mastitis, Diagnosis and Treatment" and "Hemorrhagic Septicemia," both by Dr. H. W. Jakeman, Boston, Mass. An interesting discussion followed.

Dr. L. H. Adams, B. A. I. Inspector-in-Charge, Montpelier, then gave a short resumé of tuberculosis eradication work done in the past eight years, and Hon. E. H. Jones, Commissioner of Agriculture, Montpelier, made a few brief remarks relative to the work of his department.

In the evening the members, their wives and guests enjoyed a Vermont turkey dinner, followed by entertainment and dancing.

An all-day clinic was held the following day at the small-animal hospital of Dr. Lucius D. Perry. Dr. William M. Simpson, of Malden, Mass., acted as equine clinician, and Dr. H. L. Mills, of Burlington, was in charge of the small animals.

At the business session of the meeting, the Executive Committee voted to accept the following new members: Drs. David Hopkins, Brattleboro; Albert F. Ranney, Putney, and David Walker, Woolcot. They also voted favorably on affiliation with the A. V. M. A.

The following officers were elected to serve during the coming year: President, Dr. Lucius D. Perry, Saint Albans; first vice-president, Dr. Norman H. Tenney, White River Junction; second vice-president, Dr. J. J. Staab, Montpelier; secretary-treasurer, Dr. G. N. Welch, Northfield (reëlected).

G. N. WELCH, *Secretary.*

NEVADA STATE VETERINARY ASSOCIATION

The mid-year meeting of the Nevada State Veterinary Association was held in the Agricultural Building, University of Nevada, Reno, July 20, 1932.

After the business of the meeting had been disposed of at the afternoon session, two papers were read, "Some Experiences in the Recent Outbreak of Foot-and-Mouth Disease," by Dr. R. A. Given, and "Some Sidelights on Avian Tuberculosis Eradication with Special Reference to the Extent of the Disease in Nevada," by Dr. W. F. Fisher. These were followed by a number of case reports.

At the evening session, the following papers were presented: "The Recovery of *B. Abortus* from Milk Samples Collected from Bang's Disease Reactors," by Miss Martha Huber; "The Equestrian Events of the Coming Xth Olympiad," by Dr. Warren B. Earl, and "An Experimental Study of Encephalomyelitis," by Dr. L. R. Vawter.

WARREN B. EARL, *Secretary.*

SOUTHEASTERN WISCONSIN VETERINARY ASSOCIATION

The Southeastern Wisconsin Veterinary Association met at Mayville, on August 18, 1932.

Dr. George H. Dedolph and other Dodge County veterinarians acted as hosts at an excellent dinner. Dr. J. O. McCoy then presented a paper on tetanus, after which methods of treatment for this disease were discussed. A round-table discussion on various other topics followed.

The election of officers for the ensuing year resulted as follows: President, Dr. D. E. Bleecker, Columbus; vice-president, Dr. O. Phelps, Beaver Dam; treasurer, Dr. G. A. Gettleman, Hartford (reelected); secretary, Dr. J. O. McCoy, Reeseville (reelected).

J. O. MCCOY, *Secretary.*

Cow Alarmed to Death

The nature of the mysterious malady of a cow belonging to an Iowa farmer was revealed by a postmortem examination, when the veterinarian found the material for an alarm clock in one of her stomachs. Cogwheels, springs and so forth were all present but unaccounted for.

NECROLOGY

DAVID A. BENSON

Dr. David A. Benson, formerly of Bark River, Mich., died in Rochester, Minn., July 21, 1932. He had resided in Chicago for about three years and was taken sick on Memorial Day. His condition became gradually worse and he was taken to Rochester for an operation.

Born at Iron River, Mich., August 9, 1890, Dr. Benson attended local schools and then entered the Grand Rapids Veterinary College. Following his graduation in 1918 he entered private practice at Bates, Mich. During the World War he served as a second lieutenant in the Veterinary Corps, U. S. A. After he was discharged from military service he entered practice at Bark River, where he remained until about three years ago, when he entered the service of the U. S. Bureau of Animal Industry and was assigned to meat inspection in Chicago.

Dr. Benson joined the A. V. M. A. in 1929. He was a member of the National Association of B. A. I. Veterinarians. He is survived by his widow (née Marie Lavender), two sisters and one brother. Funeral services were held at Bates under Masonic auspices.

WARREN D. LIVERMORE

Dr. Warren D. Livermore, of Aurora, Ill., died in Saint Joseph Mercy Hospital, July 22, 1932, following an operation for acute appendicitis.

Born at Pleasant Plains, Ill., October 5, 1887, Dr. Livermore attended grade school and then high school for one year, before entering the McKillip Veterinary College. He was graduated in 1917 and entered the service of the U. S. Bureau of Animal Industry shortly thereafter. For about nine years he was assigned to virus-serum control at Aurora, Ill., and Grand Island, Neb., and during recent years was on meat inspection in Chicago.

Dr. Livermore joined the A. V. M. A. in 1917. He was a member of the National Association of B. A. I. Veterinarians.

ROSCOE C. GRIFFITH

Dr. Roscoe C. Griffith, of Los Angeles, Calif., committed suicide by taking poison under rather dramatic circumstances, July 26, 1932.

Born at Saint Joseph, Mo., November 29, 1890, Dr. Griffith attended grade and high schools before entering the Indiana Veterinary College. Following his graduation in 1923, he entered general practice at Larchwood, Iowa. Later he went to California and at different times was connected with the Poso Farm, at Firebaugh, and the Arden Dairy, at El Monte.

Dr. Griffith joined the A. V. M. A. in 1924.

THOMAS JAMES TODD

Dr. T. James Todd, of Rushville, Ind., expired at his home, July 27, 1932, after a serious illness of two weeks. He had been in failing health for some time. The deceased was a native of Canada, born there May 28, 1858, and came to this country following his graduation from the Ontario Veterinary College, in 1889. He is survived by his widow, three sons, two brothers and four sisters.

CURTIS ALFRED CLARK

Dr. Curtis A. Clark, of College Corner, Ohio, died suddenly, August 14, 1932, at the home of his daughter, Mrs. James Armstrong, in Dayton, Ohio. Death was due to heart disease, with which Dr. Clark had been afflicted for several years. He was 58 years of age.

Dr. Clark was a graduate of the Indiana Veterinary College, class of 1903. He practiced at College Corner, Ohio, for almost thirty years. He joined the A. V. M. A. in 1912. His fraternal affiliations included the Masons, Knights of Pythias and Modern Woodmen. He is survived by his widow and two daughters.

Dr. George W. Roddy, of Holgate, Ohio, died June 22, 1932. He was a registered non-graduate practitioner.

Our sympathy goes out to Dr. Robert B. Grimes, of Kansas City, Kan., in the death of his wife, née Agnes Eagle, at the family home, June 29, 1932. Mrs. Grimes was a sister of three well-known veterinarians: Drs. R. Fred Eagle, of Chicago, Ill.; W. W. Eagle, of Kansas City, Kan., and T. J. Eagle, of Savannah, Mo. She is survived also by her mother, one daughter and three sons.

PERSONALS

MARRIAGES

Dr. J. E. Ingmand (Chi. '06), of Red Oak, Iowa, to Miss Ruth Hornaday, of Des Moines, Iowa, July 23, 1932.

Dr. Hartwell Robbins (K. C. V. C. '06), of Jackson, Miss., to Miss Iva Ball, of Columbia, Miss., August 6, 1932, at Columbia, Miss.

PERSONALS

Dr. Clarence H. Pals (Iowa '32) has located at Thornton, Iowa.

Dr. C. E. Juhl (Iowa '13), of Osage, Iowa, presides over his city as Mayor.

Dr. James R. Collier (O. S. U. '32) has selected Greenfield, Ind., for a location.

Dr. Elmer L. Rooks (O. S. U. '32) has decided to practice at Greenfield, Ohio.

Dr. O. F. Butterfield (Chi. '96) has removed from Gibson City, Ill., to Henry, Ill.

Dr. R. M. Parry (U. P. '30), has removed from Duncannon, Pa., to Chestertown, Md.

Dr. F. A. Anderson (Iowa '32) has located for general practice at Cedar Falls, Iowa.

Dr. J. A. Ford (Ont. '10) has removed from Long Beach, Calif., to Santa Paula, Calif.

Dr. Carlton C. Ellis (Corn. '31) recently gave a new address: Route 1, Brooktondale, N. Y.

Dr. Austin B. Byles (San. Fran. '04) is now practicing at 932 Venice Blvd., Los Angeles, Calif.

Dr. George R. Agin (Cin. '09), of Miamitown, Ohio, recently opened an office in Cheviot, Ohio.

Dr. T. A. Dermody (Iowa '32) has selected Breda, Iowa, as a suitable place for starting a practice.

Dr. Charles Webster (Colo. '14) reports a change of address from Great Bend, Kan., to Scott City, Kan.

Dr. H. H. McIntyre (Mich. '15), formerly with Merck & Co. at Rahway, N. J., is now at Enfield, N. H.

Dr. Leo P. Miller (Iowa '24), of Buffalo Center, Iowa, is a candidate for state senator on the Democratic ticket.

Dr. John H. Rust (K. S. C. '32) is associated with Dr. F. F. Russell (Ont. '13), at 286 Pleasant St., Concord, N. H.

Dr. H. G. Tully (Corn. '29) reports a change of address from Hollywood, Calif., to 1039 Grover Ave., Glendale, Calif.

Dr. Carl W. Groppe (O. S. U. '32) has opened the Glenwood Small Animal Hospital at 605 Fulton Street, Wheeling, W. Va.

Dr. Robert P. Armstrong (O. S. U. '31), formerly of Shippensburg, Pa., located at Newville, Pa., for general practice, June 1.

Dr. M. L. Claflin (McK. '17), of Neillsville, Wis., has purchased the practice of Dr. R. A. Johnston (McK. '18) at Barron, Wis.

Dr. Richard Harvey (O. S. U. '16), of Montpelier, Ohio, gets a lot of fun out of raising fancy Bermuda onions for the market.

DR. H. M. GRAY (McK. '17), of Plainfield, Ill., has purchased the practice of Dr. Lloyd D. Jones (Iowa '31), at Hampshire, Ill.

DR. F. L. MCCOLLISTER (O. S. U. '13), of Willoughby, Ohio, has purchased the Dude Ranch on the Lost Nation Road, near Willoughby.

DR. L. FINNEY (McK. '12), of Georgetown, Ill., donated his services in vaccinating 27 pigs for the Westville Community Relief Committee recently.

DR. R. G. MOORE (Iowa '13), of Dunlap, Iowa, has entered the political arena and will be a candidate for the state senate from Harrison County.

DR. D. W. GATES (U. P. '25), who has been at the New York State Veterinary College, Cornell University, the past year, is now located at Nittany, Pa.

DR. M. JACOB (U. P. '99), of Knoxville, Tenn., addressed the Knoxville Rotary Club, on July 19. His subject was "The East Tennessee Division Fair."

DR. ERNEST SCHAEFER (Chi. '17), of Bessemer, Mich., officiated as chairman of the race program for the Gogebic County Fair, held during the third week of August.

DR. W. L. BOLIN (St. Jos. '18-Ont. '21), who has been practicing at Barron, Wis., for the past two years, has entered the service of the U. S. Bureau of Animal Industry.

DR. G. S. MUIR (Ont. '28) successfully passed the recent examinations of the Royal College of Veterinary Surgeons and has been admitted as a member of that body.

DR. A. F. SCHRAGE (Chi. '11), of Plymouth, Wis., has announced his candidacy for assemblyman from the Second District of Sheboygan County, on the Democratic ticket.

DR. EDWIN C. F. ENGE (U. P. '31), who was resident surgeon at the University of Pennsylvania Veterinary Hospital the past year, entered general practice at Drexel Hill, Pa., on August 1.

DR. ALAN C. SECORD (Ont. '29), who took postgraduate work at Ohio State University the past year, opened the Secord Animal Clinic at 1164 Yonge Street, Toronto, Ontario, September 1.

DR. S. H. HOLLOWAY (Ont. '29), who has been taking postgraduate work at Ohio State University the past year, has returned to the Animal Diseases Research Institute, Hull, Quebec.

DR. F. H. KELLY (U. P. '18), formerly of Goldfield, Iowa, has joined the staff of the Ambassador Dog and Cat Hospitals, Inc., Ltd., Los Angeles, Calif. Dr. Kelly will be in charge of the department devoted to large animals.

DR. J. S. CRABTREE (Ind. '94) of Danville, Ill., narrowly escaped serious injury recently, when he was kicked on the right leg by a horse. No bones were broken, although the muscles were badly bruised and several ligaments torn.

DR. H. F. FAILOR (Ont. '11), of Spencerville, Ohio, goes in for breeding large-type Poland China hogs. He has a boar, sired by "Emblem," grand champion at the 1931 Ohio State Fair, that recently tipped the scales at 600 pounds. It will be entered in the junior yearling class at the fair this year.

DR. RUSSELL MCNELLIS (Iowa '28) has resigned his position as Superintendent of Live Stock for the United Fruit Co., Banes, Cuba, and has accepted a commission in the Veterinary Corps, U. S. Army. He is now stationed at the Army Veterinary School, Army Medical Center, Washington, D. C.